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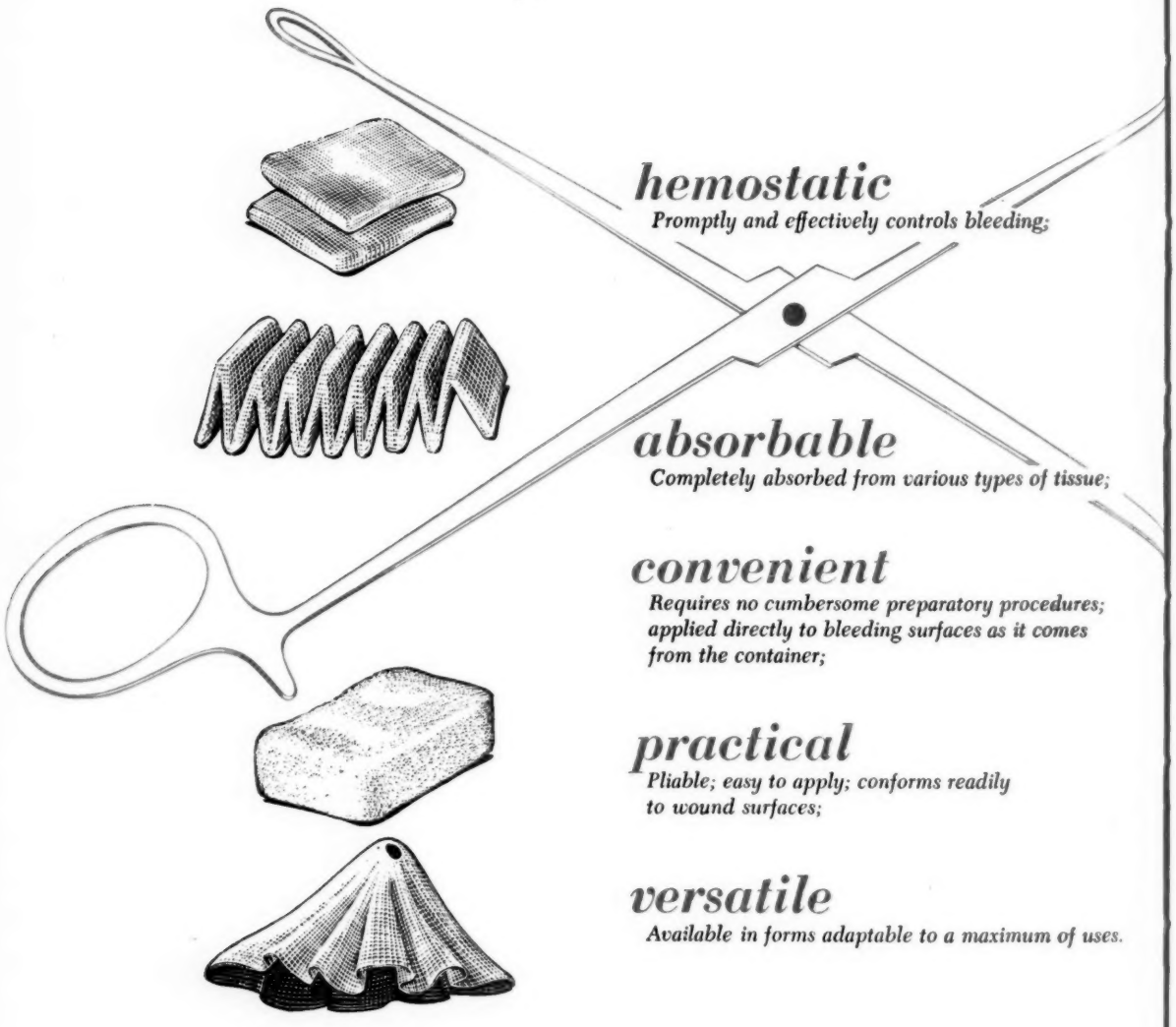

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GUEST EDITORIAL

Lower Nephron Nephrosis

THE syndrome known as lower nephron nephrosis has received considerable attention in recent medical literature. It consists of acute renal failure that often follows a state of shock and, in a large proportion of cases, progresses to uremia with a fatal termination. Numerous causes have been held responsible, including transfusions with incompatible blood, crushing injuries to muscles, operative shock, hemorrhage, burns, uteroplacental damage, and poisons, particularly sulfonamides and carbon tetrachloride.

The clinical features and pathologic changes of lower nephron nephrosis follow a rather characteristic pattern. With or without an antecedent period of shock, initial symptoms are apt to include nausea, vomiting, abdominal pain and weakness, to be followed, often insidiously, by oliguria or anuria. Later developments are rising blood pressure, increasing non protein nitrogen, acidosis and edema. In most fatal cases pulmonary edema has been found as a terminal event, while in the cases that recover, diuresis tends to appear on the fifth to the thirteenth day.[1]

The kidneys show edema with precipitation of pigment casts in convoluted and collecting tubules and degeneration of tubular epithelium. There is no significant change in the glomerular capillaries. Changes in the tubules have been noted thirty-two to seventy-six hours after onset and progress up to the fifth day or longer. Available evidence indicates that re-epithelization of the tubules, though imperfect, is completed generally within two weeks of onset. Though the persistent oliguria may be due in large part to cellular damage, there is strong evidence that functional disturbance of renal blood flow with resulting cortical ischemia plays an important role.[2]

Renal damage in lower nephron nephrosis is acute, with a very definite tendency to tissue regeneration and functional recovery. Moreover, this takes place within a period sufficiently short to permit survival even in the presence of greatly diminished kidney function. During the stage of severe oliguria, diuresis cannot be provoked by any known means and attempts to do so are apt to be harmful. Kidney decapsulation has seemed occasionally useful, but reported cases indicate that this measure has been successful only when the operation has been done after the eighth day or at a time when spontaneous recovery is to be expected. The problem then in therapy is not to produce diuresis but to keep the patient alive until some degree of healing and recovery of function occur.

It is generally agreed that the practice of flooding the body with large quantities of fluid is contra-indicated, as it does not induce diuresis and increases the likelihood of fatal pulmonary edema. Most observers believe that fluid intake orally or by infusion should be limited to the estimated loss of body fluid by vomiting and otherwise. Nutrition should be maintained by blood transfusions during the period of shock or in the presence of anemia, by adequate diet if it can be taken, and by glucose parenterally, together with water soluble vitamins. Sodium chloride is indicated if the

blood chloride is diminished and sodium lactate when evidence of acidosis appears. For delaying the accumulation of toxic excretory products, the artificial kidney or peritoneal lavage have been employed with apparently satisfactory results, but for obvious reasons these measures have not come into general use.

Hoffman and Marshall[3] have recently published an account of six cases of lower nephron nephrosis treated according to the principles outlined above with one interesting addition. Recovery in five of the six cases warrants serious consideration of the method proposed. The procedure of these workers for retarding the accumulation of toxic excretory products was the careful induction of edema of nearly normal electrolyte composition for the purpose of diluting the toxic substances, and was accomplished by additional fluid and sodium chloride. This idea is theoretically plausible and brings up the question as to whether or not the edema found in acute nephritis may be a conservative process. However, experimental observations such as those of Barry, Shafton and Ivy[4] on nephrectomized dogs are not wholly confirmative. Their findings led to the conclusion that while the giving of sodium chloride prolongs life, the production of edema is of no particular value.

Apparently lower nephron nephrosis is identical with "crush syndrome" and "traumatic uremia", previously described. From the number of cases reported, it is certain that the condition is fairly common and that it results in an extremely high mortality. Though much remains to be learned of the underlying pathological physiology, treatment based on knowledge already at hand offers considerable promise of prolonging life in many patients until spontaneous recovery takes place.

J. MORRISON HUTCHESON, M.D.

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2. Trueta, Josep, *et al.:* Studies of Renal Circulation, C. C. Thomas, Springfield, Ohio, 1947.

3. Hoffman, W. S., and Marshall, D.: Management of Lower Nephron Nephrosis, *Archives of Internal Medicine,* March, 1949.

4. Barry, Shafton and Ivy: Experimental Edema in Nephrectomized Dogs, *Archives of Internal Medicine,* February, 1933.

VASCULAR HYPERTENSION*

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and
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Etiology—DR. HORTENSTINE

Elevation of the blood pressure may be observed in a great many diseases and under many different circumstances. The normal blood pressure is 120 mm. of mercury systolic and 80 mm. of mercury diastolic with the allowable upper limits of 150 mm. systolic and 90 mm. diastolic. It should be emphasized that these limits are set for the blood pressure of a person at physical and mental rest. During severe physical exertion, the blood pressure rises precipitously and it is estimated that levels as high as 200 systolic and 110 diastolic may be reached. During our routine work, we are daily impressed with the fact that emotional upset may elevate the blood pressure and frequent readings must be made to properly evaluate many of these patients.

Elevation of the systolic pressure with a normal or low diastolic blood pressure is commonly encountered and in the majority of instances is due to arteriosclerosis, particularly that affecting the aorta and other large arteries. With the sclerotic process there is a gradual loss of the normal elasticity of the vessel wall and, instead of expanding to accommodate the influx of blood during systole, the arterial tree acts as a rigid tube and a high pressure develops. This pressure is transient, however, and it is rare that one encounters any of the stigmata of true hypertension (cardiac enlargement, cerebral accident or renal failure) in these cases.

In cases of aortic regurgitation, the drop in diastolic pressure is the most striking finding, but in many instances the systolic pressure is found to be elevated. Several factors may play a part in this phenomenon. In the first place, the wall of the aorta is usually involved in the same process which affects the valve and we have again a rigid tube substituted for the elastic tube which is normally present. Secondly, a portion of the systolic output of the heart regurgitates into the ventricle during diastole. As a result, the ventricle dilates and hypertrophies to produce a compensatory increase in the output of the heart. Because of the increased output per beat, a systolic pressure greater than normal is developed.

The increase in systolic blood pressure observed in individuals with a marked sinus bradycardia or in patients with complete heart block, is also due to an increase in the output of the heart per beat. There is an increased filling of the ventricle during the long diastolic period, followed by more complete emptying of the heart during the prolonged systole which accompanies a slow heart rate.

Hyperthyroidism is usually accompanied by an increase in the pulse pressure and a definite elevation of the systolic blood pressure is frequently encountered. There is a marked increase in the blood flow of the hyperthyroid patient which may be as much as 50% above the normal at rest. This increase in blood flow is due both to the increased pulse rate, and to the increase in the output of the heart per beat. Here again, with an increase in the volume of blood ejected from the heart during systole, there is a corresponding increase in the systolic blood pressure.

An arterio-venous aneurysm, if large, may produce a number of changes in circulatory dynamics due to the shunt of arterial blood directly into the vein. We have a situation here similar in its effects to that seen in aortic regurgitation. Because of a loss of a portion of the systolic output of the heart, there is a consequent increase both in heart rate and in the output of the heart per beat. Because of this latter finding, there may be an increase in the systolic blood pressure.

The physician daily encounters patients who may show systolic hypertension on the basis of the causes discussed above. It is important that the elevated

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systolic pressure be recognized for what it is, a useful sign indicating a derangement of circulatory dynamics. Every effort should be made to determine the underlying pathology. It should be emphasized that the elevation of the blood pressure in itself is not important, but rather that its seriousness is dependent only upon the type and extent of the primary lesion. These cases should neither be considered as true hypertension nor be so labelled.

Diastolic hypertension is very commonly encountered and in approximately 95% of the cases its cause is unknown and is so spoken of as "essential hypertension". The elevation of the diastolic blood pressure may range from 90 to 160 mm. of mercury. In the usual case, the systolic pressure will vary between 190 and 250 and on occasion reach as high as 300 mm. of mercury. The fundamental cause of this elevation is a generalized vasoconstriction with an increase in the peripheral resistance to blood flow and a consequent elevation of blood pressure, both systolic and diastolic. The effective peripheral resistance is located at arteriole level, and it is at this point that maximal vasoconstriction occurs. In its early stages, the variations in blood pressure may be transient and easily overlooked. The later course is very variable, occasionally a spontaneous cure is encountered, more commonly the blood pressure becomes fixed as is observed in the ordinary clinical case, and, finally, it may progress with remarkable rapidity to produce the so-called malignant hypertension. In the early stages there may be no apparent pathological change noted but sooner or later evidence of damage to the small arterioles can be demonstrated with subsequent involvement of the arteries. As this organic damage progresses, the narrowing of the arterioles becomes relatively fixed and the hypertension, accordingly, is irreversible. With the progress of the disease, death will eventually result, either from heart failure, renal failure or cerebral accident.

In a few cases of diastolic hypertension, the underlying cause of the vasoconstriction is known and can be recognized after careful study. In any neurological condition which is accompanied by increased intracranial pressure, there may be an associated hypertension. In this case the compression of vessels about the brain stem reduces the oxygen supply to the vasomotor center which results in a generalized vasoconstriction and elevation of the blood pressure. Similarly in certain types of adrenal tumor or hyperplasia, there may be a marked elevation of blood pressure. This is probably due to an intermittent discharge of large amounts of adrenalin into the blood stream producing a generalized vasoconstriction. As a rule, this type of hypertension occurs in definite paroxysms but on occasion a sustained elevation of blood pressure may be produced.

Hypertension may be present in almost any type of kidney disease. In some cases this may be primarily vascular, the hypertension arising from renal ischemia. The vascular lesion itself may be in the heart as in congestive heart failure, in the aorta as in coarctation, in the main renal artery as in stenosis or thrombosis, or in the renal arterioles as in periarteritis nodosa. In other cases the primary lesion involves the renal parenchyma and hypertension is commonly observed in advanced chronic glomerulonephritis, chronic pyelonephritis and in polycystic disease. Finally, the hypertension may be due to any obstruction to the outflow of urine, whether this be at urethral or ureteral level.

Regardless of the site of the lesion which is interfering with renal function, the mechanism for the production of the hypertension is probably the same and results from the elaboration by the kidney of a vasoconstrictor substance which enters the circulating blood and produces a generalized vasoconstriction and hypertension. It is conceivable that such a series of events represent an attempt on the part of the body to maintain adequate blood flow to the kidney so that the kidneys' vital function may be preserved.

As stated above where no assignable cause can be uncovered, the hypertension is spoken of as essential and these cases constitute 95% of all diastolic hypertension. There is no doubt that the hypertension is due to a generalized vasoconstriction and increase in peripheral resistance. Experimental work can be quoted to suggest either, that this is primarily renal in origin, that it is primarily adrenal in origin, or that it is primarily neurogenic in origin.

Retinal Changes—DR. MCGUIRE

A recent report by a Committee of the American Ophthalmological Society presents the most complete and, I believe, the most accurate classification of retinal lesions in the presence of vascular hypertension. There are certain points in this report which should be given general circulation, and

I will here attempt to correlate some of the more salient features of the retinal changes.

There are three types of alteration which occur in the retinal arterioles in the different phases of hypertensive disease:

(1) Generalized narrowing (representing the generally increased tone of the arteriolar system).

(2) Sclerosis (thickening of the arteriolar wall).

(3) Focal constriction (spasm).

The old, and still commonly used term "retinitis", has largely been abandoned and the term "retinopathy" substituted. This newer term means the abruptly developing involvement of the retina characterized by serous and hemorrhagic extravasations into and under the retina. The serous extravasation may be represented by diffuse edema, by localized "cottonwool" patches, and by the more chronic punctate deposits considered to be the late results of edema. For the diagnosis of retinopathy, the presence of either cottonwool patches, edema, or edema residues is necessary. Thus, if either of these conditions is present the diagnosis of retinopathy can be made even though hemorrhages are not present; however, if hemorrhages are present without evidence of any of these manifestations of retinal edema, the presence of the hemorrhages should be noted but the diagnosis of retinopathy cannot be made. When edema of the nerve head occurs in association with retinopathy the term neuroretinopathy is generally accepted. The component elements of retinopathy are basically indentical in all forms of hypertensive and renal disease. Elevation of blood pressure in itself is the most essential factor in the production of the arteriolar and retinal lesions, although constriction of the systemic arterioles in response to some circulating vasopressor substance, or to a central reflex mechanism, may be a factor in initiating the rise in blood pressure. Therefore, practically identical lesions can occur both in primary essential hypertension and in hypertension secondary to acute or chronic glomerulonephritis, toxemia of pregnancy, unilateral atrophic kidney, pyelonephritis, pituitary basophilism, adrenal tumor, periarteritis nodosa and the like.

GRADING OF LESIONS OF RETINAL ARTERIOLES

Generalized narrowing of arterioles.

Grade 1. Reduction of caliber of arterioles to ¾ average caliber or ½ caliber of veins.

Grade 2. Reduction of caliber of arterioles to ½ average caliber or ⅓ caliber of veins.

Grade 3. Reduction of caliber of arterioles to ⅓ average caliber or ¼ caliber of veins.

Grade 4. Arterioles thread-like or invisible.

Focal constriction or focal sclerosis of arterioles.

Grade 1. Localized narrowing to ⅔ caliber of proximal segment of arteriole.

Grade 2. Localized narrowing to ½ caliber of proximal segment.

Grade 3. Localized narrowing to ⅓ caliber of proximal segment.

Grade 4. Arteriole invisible beyond point of constriction (or in the case of focal sclerosis visible only as a thin, fibrous cord).

Generalized sclerosis of arterioles.

Grade 1. Brightening or increased luster of the arterioles; mild depression of the veins at the point of arteriolar crossing, with reduction in visibility of those portions of the veins which underlie the crossing arterioles.

Grade 2. Burnished coppery color of the arterioles, with definite depression of the underlying veins, widening of the apparent arteriolovenous crossing spaces and almost complete invisibility of those portions of the veins which underlie the crossing arterioles.

Grade 3. Polished silver color of the arterioles, widening of the apparent arteriolovenous crossing spaces with a change in the course of the veins ("right-angled crossings"), complete invisibility of those portions of the veins which underlie the crossing arterioles and distal dilation of the veins.

Grade 4. Arterioles visible only as fibrous cords, without blood stream.

CLASSIFICATION OF VASCULAR HYPERTENSION

Terminology	*Diagnostic Lesions in Retina*
Neurogenic Hypertension.	None, even in cases of considerable duration, except, perhaps, mild generalized narrowing of arterioles.
Acute Hypertension ("Angiospastic")	No arteriolosclerosis. Generalized narrowing of arterioles. Focal constriction of arterioles. Edema of retina, cottonwool patches, hemorrhages (usually). Edema of disk (often). May terminate in recovery, in chronic hypertension or in rapidly progressive malignant hypertension.

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Chronic Non-Progressive ("Benign") Hypertension	Only mild generalized narrowing of arterioles (Grade 1 or 2) if seen in early years. Also mild generalized arteriolosclerosis (Grade 1 or 2) if seen after several years duration of the hypertension. At times ischemic or hemorrhagic infarctions of retina.
Chronic Progresive Hypertension	Always generalized arteriolosclerosis. Generalized narrowing of arterioles. Focal constrictions in arterioles. At times focal arteriolosclerosis. At times cottonwool patches and hemorrhages in retina.
Terminal Malignant Hypertension	Always generalized arteriolosclerosis. Always papilledema. Generalized narrowing of arterioles. Focal constrictions in arterioles. At times focal arteriolosclerosis. Usually edema of retina, cottonwool patches and hemorrhages. Often residues of edema in form of macular stars or scattered punctate deposits.

Medical Treatment—DR. LACY

The medical management and treatment of essential hypertension is one of the many unsatisfactory chapters of therapeutics. The fundamental causes of essential hypertension are largely obscure and for the most part we can neither remove nor combat them. In many cases no means of treatment at our disposal will serve to lower the blood pressure for any significant length of time. In other cases the blood pressure may be lowered for a considerable period of time, but it should be remembered in such cases that we have been successful in treating the symptoms of hypertension rather than the cause of the disease. Statisticians estimate that half of the population over the age of forty will die of hypertension or its complications, and that there are 15,000,000 people with hypertension in the United States today. It is true that a certain percentage of hypertension patients will live a normal life expectancy without any apparent harmful effects from the disease. On the other hand, forty to fifty per cent of patients with essential hypertension will develop evidences of cardiac failure; somewhat less than forty per cent will manifest predominant involvement of the central nervous system; ten per cent will develop marked impairment of renal functions and die of uremia. In light of these figures, therefore, we should not assume too pessimistic an attitude toward the treatment of essential hypertension, for there can be no doubt that in many cases much can be done for the patient in the attempt to prevent various manifestations of the disease and in combating them when they arise. In most cases an attempt should be made to reduce arterial tension if this can be done by benign methods.

The success of medical treatment of essential hypertension depends upon an accurate appraisal of the type of high blood pressure present. In each patient the relative involvement of the heart, brain, or kidney or the complete absence of these manifestations must be determined. The treatment itself may be divided into (1) Psychotherapy, (2) Mental and physical rest, (3) Diet, (4) Drugs, (5) Operative procedures.

Psychotherapy—It is not meant by psychotherapy to subject every patient with essential hypertension to extensive psycho-analysis. The patient should be told as intelligently as possible the nature of his disease, and what he might expect from it. The value of the blood pressure reading should not be over emphasized, and should certainly not be made a criterion for judging improvement of the disease. It is probably wise to tell most patients what their blood pressure level is at all times. He will probably not worry as much as he would likely do if it hung as a mystery over his head. It is probably worth while to spend some time in listening to the personal troubles and difficulties of patients with essential hypertension trying to give them insight into their emotional conflicts, and in reassuring them.

Rest—Over exertion, excessive emotional strain, long hours of work, and loss of sleep aggravate essential hypertension. Rest is one of the most effective means of treating hypertension. Strain on the circulatory system and frequently the unpleasant symptoms such as headache and visual disturbances are relieved by this simple procedure. In order to achieve this, especially in very nervous patients, sedatives should be prescribed, alternating between the barbiturates and bromides. Again, this particular point may be frequently over emphasized to the extent that some patients are made to

think they are invalids, and others to worry more if they cannot find the time to rest as the physician has prescribed. The physician should be able to advise a regime which will best suit each individual patient.

When the first symptoms of cardiac insufficiency develops, such as dyspnea on exertion, orthopnea or, occasionally, paroxysmal nocturnal dyspnea, absolute bed-rest should then be insisted upon. The patient should be digitalized and in cases where chronic passive congestion is in evidence, the diuretics such as ammonium chloride and mercupurin should be used.

Diets—Generally speaking, hypertensives should not be restricted too strenuously unless obesity is present also. A patient who is over-weight should be made to reduce. In others, the diet should be moderate as to proteins, fluids and salt, but not restricted.

Certain types of diets have been used as a therapeutic measure to reduce the hypertension. Of these the most familiar is the rice diet, consisting of rice, fruit and sugar. No salt is allowed. It contains 2000 calories, 5 grams fat, 20 grams protein, derived from rice and fruit, and 468 grams of carbohydrates. Water is limited to 100 cc. daily.

The other type of diet which has been used therapeutically is the low sodium diet of Grollman. This supplies a larger amount of proteins, but the sodium intake is kept under 5 grams a day, and is accomplished by using milk, dialyzed free of sodium. This diet is, likewise, exceedingly difficult to follow, and the patients must be watched carefully for any signs of sodium deprivation.

Drugs—Edward Fries has advanced certain criteria for effectiveness of hypotensive drugs which I would like to list:

(1) That it should lower the blood pressure significantly in a fair proportion of patients.

(2) The hypotensive effect should be accomplished without serious detriment to the patient.

(3) Accompanying the hypotensive response there should be definite clinical indications of arrest, or preferably reversal of the disease, such as symptomatic relief, reduction in the cardiac size, and clearing of neuro-retinopathy.

(4) The drug should have a duration of action of at least eight hours; otherwise, it becomes necessary to administer frequent doses which interfere with the patient's normal habits, particularly with his sleep.

(5) It should be effective by mouth or at least by subcutaneous injections.

With these criteria in mind, it can be stated that such substances as extract of onions, mistletoe, garlic, and watermelon seed definitely have no efficacy in the treatment of hypertension.

A: *Nitrites and nitrates*—these substances will reduce the blood pressure in most cases of essential hypertension but their effect is so transitory that they are of little or no use in the treatment of this disease. If angina pectoris is a complication, then they are, of course, of great benefit in controlling this condition.

B: *Theobromine and other purine derivatives*—no notable effect on the blood pressure has been demonstrated by use of these drugs.

C: *Iodides*—any noticeable benefit is questionable.

D: *Thiocyanates*—because of the potentially highly toxic reaction which may result from administration of this drug, it should not be administered except in hospitals or certain clinics where accurate blood levels can be determined. The serum concentration should never exceed 12 mg. per 100 cc. The severe reactions are psychosis and exfoliative dermatitis; less toxic reactions, such as gastro-intestinal disorders, may result. The usual level of serum concentration advised is between 8 and 12 mg. per 100 cc.

E: Experimental work is being carried out in certain clinics on the so-called sympatholytic agents and their effect upon hypertension. A few of these only will be mentioned.

(1) *Tetraethylammonium bromide or chloride* (Etomon)—has been used therapeutically in certain peripheral vascular diseases, but is of little use in hypertension because its response is too brief.

(2) *Dibenamine*—a nitrogen mustard derivative and highly caustic, necessitating slow intravenous administration.

(3) *Pentaquine*—a synthentic derivative of plasmorchin—large doses are required for hypertension effect and are usually accompanied by toxic manifestations.

(4) *Dihydrogenation Alkaloids* of the ergo-toxin group—Dihydroergocornine (DHO 180)—supplies still limited to investigative work, but are the most promising of any of these agents so far.

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F: *Veratrum viride*—excellent results have been reported from the use of this drug in a large number of patients. Recent studies of its clinical and pharmacological effects indicate that the hypotensive effect may occur in sub-toxic dosage and that there are no harmful effects upon the heart or kidneys in prolonged administration. It may be given parenterally or orally. The hypotension effect usually begins in one to two hours, reaches a maximum in 4 to 6 hours, and ends in 10 to 14 hours. Nausea and vomiting are undesirable side effects, and may occur with prolonged administration. Sometimes there might be a sense of tightness in the throat, excessive salivation and occasionally there may be paresthesia about the mouth, jaw, hands and feet. These undesirable effects can be relieved by administration of atropine. The ultimate value of these newer therapeutic agents must await the test of time.

Lastly, we should, in the proper medical management of the hypertension, give symptomatic treatment when this is required. Symptomatic treatment such as the relief of a headache will help a patient, and frequently give him the impression that his blood pressure has subsided. A great deal can be accomplished by symptomatic management to make the hypertension patient more comfortable and contented.

Surgical Treatment—DR. CUSTER

In concluding this discussion, I would like to do two things:

First—to mention one or two points in connection with the anatomy of the sympathetic nervous system, and to name the three operations which are principally employed.

Second—to summarize the results which are being reported following the use of one of these procedures, namely, the one which is designed for the control of the problem which we have for discussion tonight—high blood pressure—the so-called hypertension operation.

There are three operations which are performed so much more commonly than all others, that they may be considered for practical purposes as comprising the bulk, if not the entirety, of sympathetic system surgery.

1. There is one operation which is performed for the purpose of denervating the upper extremity —thoracic sympathectomy.

2. There is one operation which is performed for the purpose of denervating the lower extremity —lumbar sympathectomy.

3. The third operation is performed for the relief of hypertension and is known as thoracolumbar sympathectomy.

In performing thoracic sympathectomy, which incidentally is the most difficult of the three procedures technically, a so-called preganglionic resection of the second thoracic ganglion is performed for the relief of vasospastic disease of the upper extremity, principally for Raynaud's disease.

Lumbar sympathectomy is performed through a flank incision, and includes the resection of either two or three of the lumbar ganglia with the intervening segments of the chain. It is by far the easiest of the three procedures, and usually requires about 45 minutes or less from skin to skin. It is performed most commonly for the relief of vasospastic disease of the lower extremity, principally for Buerger's disease.

Thoracolumbar sympathectomy lies midway between the other two procedures in regard to technical difficulty, but is complicated by the fact that it must always be performed as a bilateral procedure, necessitating two separate major operations. In the Smithwick type of procedure, resection includes the sympathetic chain usually from the level of T-8 to L-2, plus the three splanchnic nerves.

Now, in turning to the surgical management of hypertension, I would like to refer for a moment to the historical development of the operative treatment. In 1925 Adson and Rowntree in Rochester, Minnesota, conceived the idea that a denervation of the splanchnic area might provide a bed of dilated vessels of small and medium calibre, thus creating a reserve pool to serve as a safety valve for the protection of more important vessels, principally those of the retina and brain.

Adson tried several tentative procedures, but it was Craig, also of the Mayo Clinic, who worked out the first really practical operation. In 1934 Craig devised a procedure for the resection of the lumbar sympathetic chain together with the greater splanchnic nerve and the celiac ganglia which was performed below the diaphragm and is known as the Craig-Adson operation, or subdiaphragmatic splanchnicectomy.

Then, in 1935, Dr. Max Peet, at the University of Michigan, conceived of and executed a more extensive resection performed above the diaphragm.

This supradiaphragmatic approach necessitated the resection of the 12th rib, but permitted removal of the lower five thoracic ganglia and all three splanchnic nerves.

And then, in 1940, Reginald Smithwick described an operation which is a combination of the Craig and Peet operations, and is known as thoracolumbar sympathectomy. This procedure, modified in various ways, is the one most commonly employed today.

There is a tendency toward more and more extensive resection. The leading voice in this trend is certainly that of Dr. Keith Grimson at Duke University, who is at the present time performing what amounts to practically a total sympathectomy on both sides. The results are not as yet available, and it will probably be several more years before we know whether this extensive type of surgery is or is not justified.

In surveying the surgical results, I think that there are two fundamental assumptions which we can make at the start which will help us to reduce the appraisal to a ground level from which we can secure a better take off.

The first is that it is obvious immediately that sympathectomy is *not* the ultimate answer to the hypertension problem. We all believe that the final answer lies in the direction of medical research, and that, when it comes, no surgical procedure of the magnitude of sympathectomy can, nor should, compete with it. But we also know that the solution may not be forthcoming in the near future, and perhaps not within this generation. Until it does, it would appear that surgery offers far and away the best means of control for the selected case.

The second assumption which I would like to make is that sympathectomy *is* an effective means of reducing blood pressure in most hypertensive patients. The figures which have most impressed me are the following:

In 1939, in the *American Journal of Medical Science,* Keith, Wagoner and Barker published a follow-up study of a very large series of hypertensive patients treated medically. Included in this series were some hundreds of patients who suffered from so-called "malignant hypertension", a form of the disease developing in young people and characterized by rapid progression, severe neuroretinopathy and one or more diopters of papilledema. They found that in this group 80% were dead at the end of one, 90% at the end of two years, and after five years the mortality was 99% if not 100%.

In Max Peet's series, there were included 112 cases of this same disease. The significant fact is that at the end of five years 19% were alive and reasonably well.

The best follow-up study following sympathectomy is that of Max Peet, because it is the largest. His cases have all been carefully followed from five to eleven years from the time of surgery. And I ask you to bear in mind the fact that these are cases subjected to what most surgeons now regard as being a limited operation.

These are his figures:

There were a total of 437 patients in the series (and incidentaly 82% had already progressed to serious organic disease).

Of the 437 patients, 57% were alive at the time of follow-up and 42% had died.

Of the 52% survivors 81% were considered as having attained a satisfactory result, 20% or so as having been failures. Or, expressing the results in terms of the original 437, not quite one-half obtained satisfactory five year results, the other half failing to survive or remaining unimproved.

If the hypertensive patient is under 45 with a reasonably decent cardiac and renal reserve, and has a fixed type of hypertension, yet one which will, upon the application of bed rest, or the administration of sodium amytal or tetraethyl ammonium chloride, show sufficient release to approach normal systolic and diastolic levels, it may be said that sympathectomy, while not the final answer, is probably the best answer we have at this time. Then we should acquaint that patient with the facts of his disease, and we may with confidence recommend surgery to him as offering him his best chance of remaining alive and of remaining well five years hence.

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DIAGNOSIS AND TREATMENT OF CARPAL INJURIES*

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Injuries to the carpal bones fortunately occur in only a small portion of the fracture cases treated at the University of Virginia Hospital.

We use the term "fortunately" because these injuries are most difficult to recognize and treat. Indeed, more than one-third of the cases referred to us represented injuries either misdiagnosed or entirely overlooked.

The carpal bones most commonly involved in injuries about the wrist are the navicular and the semilunar. Our discussion for the most part will be limited to injuries of these two bones.

PATHOLOGY OF NAVICULAR FRACTURES

Most of the fractures are transverse, usually in the long axis of the bone. All fractures of the body are intra-articular and are rarely displaced except when complicated by an anterior dislocation of the lunate. Few of the fractures are comminuted. Circulation is poor in the proximal portion, fair in the middle portion, and good in the distal portion.

DIAGNOSIS

There is considerable pain on the radial side of the wrist. The grip is weak and painful, and swelling is variable. Pain is more acute in the anatomical snuff box. The patient may venture his own diagnosis of severe sprain. Clinical diagnosis must in all cases be confirmed by roentgenograms. X-ray examination of the injured wrist is often referred to as a "simple procedure". It is common practice to depend on a posterior-anterior and a lateral projection to visualize the bones of the wrist. Since the carpus is part of the wrist, this examination is actually far from simple. It requires multiple projections and is frequently misinterpreted. What may, on physical examination, be taken to represent injury of the distal and of the radius or ulna, may instead be due to injury of a carpal bone. Also carpal injury may accompany injury of the distal end of the radius or ulna.

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The anatomy of the carpus is so complicated that a posterior-anterior and a lateral projection do not visualize the bones completely. Oblique projections with the hand in semi-pronation and semi-supination are essential. For example, transverse linear fractures of the scaphoid are often visible only in semi-pronation because the forward tilt of the bone hides the fracture line in the anterior-posterior and lateral projections. (Figure 1.)

Fig. 1.—Complete transverse scaphoid fracture, visible in semi-pronation only.

No x-ray examination for suspected injury of the wrist is complete without examination of the carpus. Examination of the carpus requires a minimum of four projections.

TREATMENT

Displaced fractures should be treated by traction and immobilization. All fractures of the scaphoid must be immobilized in a snug plaster of paris cast which extends from the elbow to the metacarpal heads and must include the proximal phalanx of the thumb. The wrist should be moderately dorsiflexed and in slight radial deviation. The thumb should be slightly abducted and extended. Healing usually occurs in from seven to ten weeks. The

Fig. 2A.—Examination immediately after injury. No abnormality demonstrated.

cast should be changed if it becomes soft and loose. Immobilization must be maintained until there is x-ray evidence of union. One should not hesitate to prolong immobilization for as long as six months. The treatment of non-unions and delayed unions is beyond the scope of this paper.

CLASSIFICATION AND PATHOLOGY OF SEMILUNAR INJURIES

The first type is the perilunar dislocation. This injury is one in which the semilunar is in relatively normal position and the distal row of carpals is displaced, usually in a backward and upward direction. The second type is the rotation dislocation of the semilunar, most often toward the palm. As the lunate becomes displaced, there is usually tearing of its ligament. When the anterior ligament is

Fig. 2B.—Same case, two weeks later. Fracture scaphoid clearly visible.

intact, it rotates the bone forward and upward. Rotation may vary from 90 to 250 degrees.

DIAGNOSIS

The clinical findings should suggest a diagnosis in over 75 per cent of the cases; however, the final diagnosis must be made by x-ray. True laterals and anterior-posterior views are needed. When in doubt it is useful to take plates of the normal wrist for comparison.

There is an altered contour at the anterior sur-

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face of the wrist, the fingers are maintained in slight flexion and the dislocated lunate may be palpated anteriorly. Motion of the wrist is painful. There is a variable amount of swelling in the wrist and fingers, and there may be median nerve involvement.

TREATMENT

Immediate manipulation is the treatment of choice, though reduction should be tried even in cases 10 to 14 days old. For the perilunar dislocation, maneuvers similar to those used for reduction of a Colles' fracture are suggested. A cast or anterior splint is applied from the elbow to the metacarpal head with the wrist in 30 to 50 degrees flexion. After four weeks of immobilization in flexion it is wise to change the position of the wrist to either 5 degrees of flexion or neutral, and immobilize for another 14 days.

The manipulative reduction of the fresh palmar semilunar displacement is a fairly standardized procedure which is adequately described in all of the acceptable fracture texts.

There are other methods of reduction which, in the hands of many operators, have given good results. We would warn against the so-called "broomstick" method which utilizes a broomstick rolled over the anterior surface of the wrist while traction and extension is made on the hand. Damage to the median nerve is not uncommon following this method.

Fig. 3A.—Fresh palmar dislocation of lunate.

Fig. 3B.—Same case. Successful reduction.

X-rays should be taken at intervals of four weeks for a period of at least four months in order to rule out the presence of aseptic necrosis of the semilunar. While it is true that aseptic necrosis does not usually follow simple dislocations, but does occur after crushing injuries of the semilunar, this possibility should not be overlooked, especially for medical-legal purposes.

COMPLICATIONS

Delayed union, non-union, and aseptic necrosis following fractures of the scaphoid may be due to damage to the meager circulation. It can be stated with certainty that most of the complications arise either from failure to treat the fracture through lack of recognition or from inadequate immobilization. The wrist may be persistently painful, the grip weakened, and there may be a variable amount of traumatic arthritis in the radio-carpal area.

Aseptic necrosis of the semilunar often follows a crushing type of injury. Rough manipulation can be a causative factor. Delayed reduction may lead to permanent stiffness, fixed displacement, loss of motion in the wrist and fingers, and severe traumatic arthritis. Operative interference may be required because of failure of early adequate therapy.

SUMMARY

We have attempted to emphasize in this paper the hazards to function that accompany fractures of the carpal bones, particularly the navicular and semilunar. Early recognition depends on clinical acumen and accurate x-rays. A persistently painful wrist should be re-x-rayed in ten days despite primary negative findings. One should not hesitate when in doubt to x-ray the normal wrist. Many congenital abnormalies, among them the bipartate navicular, may be picked up in this way. Many textbooks fail to include perilunar dislocations of the wrist under their classification of lunate injuries. This injury is frequently mal-treated because it is unrecognized. Somewhat different reductive maneuvers are necessary in the treatment of this injury. The maneuvers necessary for the volar dislocations are not applicable to the perilunar injuries.

Immobilization of fractures of the navicular should be prolonged until there is union or little hope of union. So called "chronic sprains" of the wrist should create suspicion of the possibility of an overlooked navicular fracture. A persistently painful wrist with pain in the anatomical snuff box should have x-rays repeated in ten days despite a primary negative plate. It frequently requires that length of time for the fracture line to become visible.

DISCUSSION

DR. GEORGE COOPER, JR., Charlottesville: I should like simply to emphasize from the radiological standpoint that the important thing about carpal injuries is their recognition. The treatment was fully discussed by Dr. Frankel. We find that most of the difficulties in carpal injuries come from failure of recognition, and we find that those failures are due to incomplete radiological examination.

DR. GEORGE A. DUNCAN, Norfolk: While there is nothing new or startling in this paper by Drs. Cooper and Frankel, it does contain truths that bear repetition time and time again. Fractures of the carpal scaphoid and dislocations of the carpal semilunar are too frequently overlooked by not only the physician but also by the roentgenologist. The latter frequently does not have the history or the physical examination for his guidance, as the patient may have been seen only by the technician. Sprains of the wrist are rare. All injured wrists, therefore, should have X-ray examination, and if symptoms persist over seven to ten days another X-ray should be made. Time lost in making the diagnosis delays the proper treatment, and a poor result may be the outcome. Failure of diagnosis naturally may lead to legal responsibilities, as can be readily appreciated.

X-rays of the wrist should be made routinely in four planes, namely, antero-posterior, lateral, and right and left oblique. If there is any question at any time the opposite member should be X-rayed for comparison.

Most fractures of the carpal scaphoid will heal if treated early by reduction and a plaster cast, to include the thumb in abduction for eight to sixteen weeks. Most first- and second-degree dislocations of the carpal semilunar can be reduced by manipulation, whereas most third-degree dislocations must be removed surgically.

This is a timely paper and should be reread by all of us from time to time to remind us of our responsibilities.

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THE INJECTION TREATMENT OF INTERNAL HEMORRHOIDS*

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Although the treatment of internal hemorrhoids by the injection of sclerosing agents has been practiced for more than seventy-five years[1] and is well established as a procedure of merit, there persists, nevertheless, in the minds of many physicians, numerous misconceptions in regard to it which we shall attempt to correct.

The idea that the injection treatment is intended to "slough off" hemorrhoids is still prevalent and doubtless stems from its early days when the method was practiced almost exclusively by ignorant quacks who did indeed have this as their aim. They were unfamiliar with the use of the speculum and could treat only those hemorrhoids prolapsing outside of the body. As might be expected, the slough did not always stop with the destruction of the internal hemorrhoids, but, beyond any doubt, all too frequently extended into adjacent structures such as external hemorrhoids, sphincter muscles, and even the genito-urinary apparatus. The frightful pain and permanent deformities following such complications, together with the reprehensible exploitation of the pile sufferer by the charlatan, created within the ethical profession a so violent antagonism to the injection method as to greatly delay the widespread acceptance and use of the procedure.

To E. H. Terrell must go the credit[2] for lifting this most valuable method of therapy from its unsavory origins and placing it on a sound scientific basis, acceptable to the most scrupulously ethical proctologist, as well as for making it entirely safe for the patient. He did this by introducing in 1916 a new drug, quinine urea hydrochloride, for the injection of hemorrhoids. He reported[3] on his three years experience with it in a series of 128 cases, and clearly and accurately outlined the indications and contraindications for treatment as well as the technique, which prior to this had been a closely guarded secret. There were some eminent proctologists who resolutely refused to concede any merit to the injection treatment, but the staunch support by colleagues of more open mind and greater vision, as well as the numerous articles on the subject by E. H. Terrell[4,5,6,7] have done much to advance the method and remove the stigma cast upon it by the quack.

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INCIDENCE AND ETIOLOGY

The true incidence of hemorrhoids in the general population is unknown, but it is high and of approximately equal distribution in the two sexes. Authorities do not agree as to the etiology, but the more we have studied the problem the more firmly are we convinced of the fallacy of the popular notion that "piles are caused by constipation". Certain it is that we see chronically costive patients without hemorrhoids, just as some of the largest hemorrhoids are seen in those to whom constipation is unknown. The real cause is probably a combination of heredity, the mechanical factors incident to the erect posture, and the results of inflammation. Of course, when hemorrhoids are present their symptoms are frequently aggravated by either constipation or the use of laxatives.

SYMPTOMATOLOGY

Usually the first symptom of internal hemorrhoids is bright red, fresh bleeding at stool. This is sometimes seen on the toilet paper in cleaning the parts, but more commonly is observed to color the water in the commode. It is not unusual for patients to report liquid blood dripping or even squirting from the anus at defecation. The passage of dark or clotted blood should make the physician suspect that its source is a lesion higher up in the bowel. In many cases the amount of blood lost is not great, whereas in others it is definitely in excess of blood production and thus produces a secondary anemia. In rare instances we encounter a patient almost exsanguinated by "bleeding piles" with hemoglobin determinations below twenty per cent. Because internal hemorrhoids are completely painless, the doctor is sometimes not

consulted until the anemia itself is severe enough to give symptoms.

The second symptom of internal hemorrhoids is protrusion; this, in the beginning, is usually during defecation and afterwards will replace itself, especially if the patient will lie down for a short while. As the condition progresses, protrusion may occur on exertion such as lifting, or when one assumes a stooping or squatting position, and will require digital replacement. If still untreated, eventually the time is apt to come when the hemorrhoids protrude and the patient is unable to replace them; this complication is known as strangulation and is usually accompanied by thrombosis and often necrosis as well. Other symptoms of internal hemorrhoids are variable and may consist of vague aching pains in the back and lower extremities, irritability and easy fatigability, or symptoms may be lacking altogether.

CHOICE OF SCLEROSING AGENT

There are available many sclerosing chemicals which have been advocated for use in the injection treatment of hemorrhoids. Phenol in a vegetable oil is perhaps the oldest agent still in use and it is effective, though it does have certain disadvantages such as the occasional formation of oleomas, production of more pain, and a greater tendency to produce slough. Our experience has been largely limited to quinine urea hydrochloride which we have used daily for many years. For the past eighteen months we have used a preparation made by the Acme Scientific Co., of Richmond, Va., and marketed under the trade name of "Quinuride", which is a 4.5 per cent solution of anhydrous quinine urea hydrochloride adjusted to a pH of 2.6. We consider Quinuride to be more efficient than any preparation we have previously used and equally as safe. The results obtained have been highly satisfactory and free from complications.

OBJECTIVE AND SUBJECTIVE RESULTS OBTAINED

To properly understand the injection treatment one must keep in mind the fact that internal hemorrhoids are tumor masses within the lower rectum composed of varicose veins. Not infrequently these are seen as enormous masses protruding through the anus and on the outside of the body. Regardless of location, if they are covered by mucous membrane instead of skin, they are properly classified as internal hemorrhoids. Varicose veins are elongated, dilated, thin walled, and tortuous, with varying degrees of phlebitis present. The tissues drained by such veins suffer from the effects of chronic passive congestion with loss of elasticity and increased fragility, leading to surface erosion and ulceration.

When a small amount of "Quinuride" is injected into an internal hemorrhoid a fibrous tissue infiltration follows which produces shrinkage of the hemorrhoidal mass. By making repeated small injections at intervals the protologist can observe the hemorrhoid becoming progressively smaller until it is finally obliterated completely. In the vast majority of cases symptoms will cease after the first treatment, and if the patient is then discharged, a recurrence should be expected. Therefore, it is important to continue until the physician is satisfied that the hemorrhoidal mass has actually disappeared. A slough can easily be produced by injecting an excessive amount of the solution at one time or by placing the solution too close to the mucous surface; however, a skilled protologist will have little difficulty in preventing this undesired complication.

PROCEDURE AND PLAN OF TREATMENT

The injection of internal hemorrhoids is an ambulatory treatment and can be safely carried out in the doctor's office without anesthesia. No particular preparation of the patient is necessary, though the rectum should be emptied by normal bowel movement. Treatments are preferably given at intervals of one to two weeks; the average case requiring four to eight injections. The treatments are practically painless, though most patients experience an uncomfortable sensation in the ano-rectal area usually beginning after thirty minutes and lasting about an hour. The patient is advised to stay off of his feet during this period after which he may resume his normal activities except that defecation should be deferred for 12 to 24 hours. When internal hemorrhoids prolapse easily a "T-binder" is applied after the injection is made to support the hemorrhoids within the rectum and thus prevent the possibility of strangulation.

SELECTION OF CASES

Most physicians today regard the injection treatment as useful to gain palliative and temporary relief, but consider surgery the only means of securing a lasting cure. It is our experience that the

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injection treatment not only achieves symptomatic relief, but that its use in cases of uncomplicated internal hemorrhoids can be expected to effect a cure as complete and permanent as that obtained by surgery. It must be understood that all types of hemorrhoids are not suitable for the injection method, and an experienced proctologist can usually determine quite accurately by careful history and examination on the first visit what results may be expected from treatment. Surgery is generally indicated when internal hemorrhoids coexist with external hemorrhoids, fissure, fistula, or polypi, but the age and general condition of the patient also must be considered in selecting appropriate treatment. With senile patients or those having serious constitutional diseases palliative relief obtainable by injections may often be wiser than attempts at radical cure by surgery. Likewise, in those who have developed an anemia secondary to bleeding hemorrhoids, it is frequently advisable to arrest the blood loss by a few injection treatments, deferring surgery until the patient's blood count and general condition are satisfactory. Thus it is important that the proctologist be competent to administer the injection treatment, or else he must deny some of his patients the relief that can so easily be secured by a few injections.

We see a large number of patients for whom surgery is indicated, but for one reason or another wish it deferred. Sometimes this is advisable because of a temporary condition such as pregnancy or convalescence from surgery or other serious illness, and sometimes it may be justified by the patient's convenience, as in those whose work is seasonal or for whom a hospital bed cannot be found. Certainly these patients will be grateful for any relief obtained, and we are careful to explain to them that though great relief is to be expected, it will be neither complete nor permanent. We must admit that some of these patients are so pleased with the results obtained by a few treatments that they abandon for the present at least their plans for surgery.

COMPLICATIONS

Except for a very rare reaction due to quinine sensitivity we have observed no complications following the injection of internal hemorrhoids with "Quinuride". We attribute the complications of abscess, slough, fistula, hemorrhage, or stricture occasionally reported in the literature to errors of technique or faulty judgment in the selection of cases. Recurrence in properly selected cases is usually due to inadequate treatment.

SUMMARY

(1) An attempt has been made to correct certain misconceptions concerning the mode and effectiveness of the injection treatment of internal hemorrhoids.

(2) The etiology and symptomatology have been discussed.

(3) The plan and procedure of the injection method have been discussed, including the choice of sclerosing agents.

(4) The criteria for the selection of cases have been outlined with the results to be expected following adequate injection therapy.

(5) Our experience leads us to conclude that quinine urea hydrochloride (Quinuride) is an effective and safe agent for use in the injection treatment of internal hemorrhoids.

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DISCUSSION

DR. T. NEILL BARNETT, Richmond: Someone has defined a consultant as one who makes a rectal examination. This should be amended to apply to one who makes an intelligent and complete rectal examination, not just a digital examination.

The number of patients that consult the gastroenterologist and proctologist with comparatively minor findings, other than a moderate to severe secondary anemia, is significant. Many are treated *ad lib.* and *ad infinitum* with little or no lasting benefit.

To cite a case, a man whose work necessitated his being on his feet most of his working hours complained of profound weakness and fatigue. The various examinations revealed a severe secondary anemia. Sigmoidoscopic examination was negative. When the anoscope was inserted, however, numerous bleeding internal hemorrhoids were revealed. On repeated questioning no history of passing blood could be obtained until after the examination, when his attention was directed to observing his stool. He indicated that the bleeding resulted from the examination but finally admitted that he had always flushed the

toilet immediately, without observation. This is a practice common to many, and it denies the physician a most helpful observation. Injection of the hemorrhoids cured the anemia and the hemorrhoids.

Constipation is certainly not a primary etiological factor but is doubtless a contributory factor. Particularly is this true of the hard, dry fecal mass that is retained in the rectum for several days. On the other hand, many patients who are constipated are greatly relieved or cured of their constipation after adequate injection of a large, prolapsing hemorrhoid that obstructs the anal canal.

Another very disturbing condition that is frequently cured by injection of quinine urea is first-degree rectal prolapse. Systematic injections around the lumen into the submucosal tissue seals the mucous membrane to the rectal wall by means of the fibrosis resulting from the sclerosing agent. This method is most effectual in the adult and is especially indicated in infancy and childhood, since it is practically free of discomfort.

Every patient with rectal bleeding, even though hemorrhoids are apparently the sole source of the blood, should be sigmoidoscoped. All too frequently a bleeding or nonbleeding polyp or cancer may be found in the same individual. It should be remembered that the polyp or small adenoma of today may be and frequently is the cancer of tomorrow.

The problem of quinine sensitivity, although rare, can be most distressing and serious. I recall one patient in whom its occurrence was almost fatal. I have adopted the plan of obtaining from the patient a history of any possible quinine sensitivity before making an injection of quinine urea. If there is the slightest doubt, or if the patient cannot recall ever having taken quinine, I give him four three-grain capsules of quinine hydrochloride to take, one every four hours, with instructions to stop the drug if there is any indication of a skin rash or other reaction.

This able presentation is most instructive and timely. I do not know of any other procedure in medicine that is so simple, yet results in such radical cure.

Radioisotope Aids Brain Surgery.

Difficult surgery for brain tumors is being made easier by radioactive phosphorus produced in atomic energy laboratories.

Writing in the May 21st *Journal of the American Medical Association,* B. Selverstone, M.D., A. K. Solomon, M.D., and W. H. Sweet, M.D., Boston, say that in 14 cases they were able to locate brain tumors at the time of operation by use of the isotope.

When radioactive phosphorus was given to these patients in injections, it became concentrated in the brain tumors. The doctors were then able to locate the tumors by using as a probe a miniature model of the Geiger-Muller counter, an instrument that measures radioactivity. The precise location of the tumor was shown by an increase in the counting rate of the instrument.

Drugs for High Blood Pressure.

Clinical testing of two synthetic compounds, priscol and dihydroergocornine, for usefulness against high blood pressure is reported in the May 21st *Journal of the American Medical Association.*

The drugs counteract constriction of the blood vessels and other effects produced by the sympathetic nerves, which regulate the heart beat, blood flow, and many other body functions.

Use of priscol in two cases of high blood pressure is described by Max P. Rogers, M.D., of High Point, N. C. In both patients, one of whom had received priscol only two weeks, blood pressure was greatly lowered, Dr. Rogers says. Both patients tolerated the drug well.

Priscol shows promise of being useful in disorders in which the blood circulation to the legs is impaired, Dr. Rodgers' study shows. Three cases of such disorders associated with hardening of the arteries, three cases associated with diabetes, and two cases associated with thrombophlebitis (clots in the blood and inflammation of the veins) showed improvement after priscol therapy.

Patients with certain heart conditions may not be able to take the drug or may be able to take it only in small quantities, however, he indicates.

Tests of dihydroergocornine against high blood pressure are reported by Drs. Robert W. Wilkins, Edward D. Freis, and Joseph R. Stanton, from the Evans Memorial and Massachusetts Memorial Hospitals, and the Department of Medicine, Boston University School of Medicine, Boston, in another article in The Journal. The activity of dihydroergocornine in lowering blood pressure is unpredictable, being negligible in some patients and "profound" in others.

XUM

ACUTE THROMBOCYTOPENIC PURPURA FOLLOWING MASSIVE HORMONE THERAPY IN A PREGNANT DIABETIC*

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This is a case report of a 20 year old pregnant white female with moderately severe diabetes. The diabetes was treated and controlled, and the patient was given stilbestrol and progesterone to try to prevent any accident during the pregnancy.

The patient was admitted to the hospital on July 23, 1947, for the control of her diabetes and at that time was approximately two months' pregnant. The diabetes was first discovered in March 1947 but had never been regulated. Her blood sugar was 238 mgm., and the urine contained 4+ sugar. She was placed on a 1650 calorie diet, C 200, P 100, F 50, 40 units of protamine zinc insulin before breakfast, and regular insulin according to test before each meal, which was approximately 20 units per day.

While the patient was in the hospital, she developed uterine cramps. She was started on 0.5 mgm. stilbestrol and 5 mgm. pranone daily, and this relieved her symptoms.

The pregnancy was uneventful until October 1947, at which time she began having severe uterine cramps. During the eighteenth week of the pregnancy, the stilbestrol was increased to 5 mgm. daily, and the dosage of pranone remained the same. The increase in the stilbestrol relieved the uterine cramps. The diet was increased during the twenty-second week to 1760 calories, and the protamine zinc insulin to 50 units before breakfast, the regular insulin remaining approximately the same.

During the thirty-second week the patient began to show signs of toxemia of pregnancy and an increase in the uterine contractions. She was treated for the toxemia of pregnancy, and the stilbestrol was increased to 15 mgm. a day. The insulin requirement remained the same.

The toxemia increased with continuous headaches and intermittent uterine cramps. The patient was admitted to the hospital the thirty-fifth week for the interruption of the pregnancy. At that time the fetal heart sound was in the left lower quadrant, 120 per minute, vertex presenting but not engaged, and the cervix was 1 cm. dilated.

*Read at the annual meeting of the Medical Society of Virginia in Richmond, October 18-20, 1948.

The diabetes was regulated, and she was given vitamin K, 15 mgm. daily for two days before the Caesarean section. The blood count on February 2, 1948, was RBC 4,010,000, WBC 6,000, hemoglobin 13½ grams, 72% polys, 28% lymphs. The blood sugar estimation was 193 mgm. and the blood pressure was 160/100.

The Caesarean section was done under spinal anesthesia, and an apparently normal male infant weighing 8 lbs. 6 oz. was delivered. At the time of the operation, she lost approximately 100 cc. of blood. Her condition remained normal with slight uterine bleeding for the next hour and a half. Suddenly she began profuse uterine bleeding, hemorrhaging from the entire length of the abdominal incision, hemorrhaging from the gums, and cutaneous manifestations of purpura especially at the site of the infusion injection. The patient went into shock immediately. The blood pressure was 70/? She was given 1000 cc. of plasma and 500 cc. of blood immediately. The blood pressure rose at 130/70, and the bleeding checked slightly. She was given vitamin K, coagulin, koagmin, vitamin C, with rutin, and 1500 cc. of whole blood during the next five hours. At the end of six hours the bleeding had checked from the uterus, abdominal incision, and gums, and the blood pressure remained normal.

The platelet count at the time of the massive bleeding was 92,400 per cubic millimeter, and the hemoglobin was 9 grams. There was no coagulation for the prothrombin. Six hours later the prothrombin time was 17 seconds, or 40% of normal.

It was noted following this, upon questioning the patient, that she had developed rather marked bleeding of the gums prior to admission to the hospital (but this had not been reported to me). The bleeding from the gums subsided somewhat after she had been given vitamin K before the delivery.

After we were able to control the uterine and incisional bleeding, the patient's blood pressure remained normal, and her course was uneventful. She was given two transfusions following this, making a total of six transfusions. The incision

healed by primary union, and she was discharged on the ninth postoperative day. The platelet count was 160,000 per cubic millimeter, RBC 3,990,000, WBC 10,900, hemoglobin 13½ grams, and blood sugar 188 mgm. She was placed on a diet of 225 grams of carbohydrates, 90 grams of proteins, 75 grams of fats, 45 units of protamine zinc and 15 units of regular insulin before breakfast daily.

The hemorrhagic diathesis which appeared in acute and alarming form after her delivery was most likely, judging from her blood work, a secondary thrombocytopenia purpura from the prolonged use of estrogenic hormones (stilbestrol) during her pregnancy.

The underlying disturbance in clotting seems to be the lowered platelet count. The bleeding tendency cleared fairly promptly following the omission of the estrogens and the multiple transfusions. It is hard to say what part the vitamin K, coagulin, koagmin, and vitamin C with rutin played in the correction of the hemorrhagic diathesis except as supportive measures.

The literature indicates that the administration of estrogenic substances and particularly stilbestrol, while at times productive of nausea and vomiting, does not cause serious toxicity in human beings. Nevertheless, certain cases have been reported, which indicates that occasional individuals may exhibit an idiosyncrasy or sensitivity to stilbestrol or other estrogenic substances with resultant injury of the liver or bone marrow.[1] Loftes[2] reported the case of a 44 year old white woman who developed purpura on the legs and bleeding from the gums from weekly injectons of an estrogen.

Watson, Schultz, and Wikoff[1] reported five cases in which purpura was first noted after estrogen therapy. Thrombocytopenic purpura has been produced in dogs with large amounts of stilbestrol.[3,4]

SUMMARY AND CONCLUSIONS

1. Case report of a 20 year old pregnant white female with moderately severe diabetes. The patient was treated with stilbestrol and progesterone to try to prevent any accident of pregnancy.[5]

2. Following the interruption of the pregnancy at the thirty-fifth week, there was an acute hemorrhagic diathesis, which was relieved by multiple transfusions.

3. It is thought that the purpura in humans is the result of a sensitivity to the estrogenic drugs.

4. It seems worthwhile that any doctor who might be giving massive doses of stilbestrol to a pregnant diabetic should be on the watch for postpartum hemorrhage, as this is a very severe complication during any delivery, whether normal or by Caesarean section.

5. If at all possible, a blood gonadotropin determination should be done, as this is a guidance to the hormone imbalance. No facilities were available in this city at the time of this case for this determination.

NOTE: The diabetes was treated and controlled by Dr. Earle Glendy, Roanoke, Va.

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DISCUSSION

DR. WILLIAM R. JORDAN, Richmond: This case of Dr. Ridgway's is certainly a very interesting one. I think it is well worth while reporting this or any others similar to it. Dr. Ridgway has discussed one of the preparations most widely used in the United States today. It is used in the menopause, in obstetrics, in bone pathology, and in the pregnant diabetic with toxemia.

You could tell from Dr. Ridgway's description that the case was well handled from every point of view, even after the trouble originated following delivery. I do not know just what else could have been done.

Stilbestrol, which is usually very mildly toxic or not toxic at all, occasionally in massive doses becomes really harmful. In the work on dogs it was shown that mild doses did not cause any damage at all, whereas massive repeated doses produced liver damage, with fatty necrosis. In humans it seems to me that it is only persons with an idiosyncrasy to the drug or who have no need for it who are harmed by it. I wrote Dr. White who is with Dr. Joslin, who has had more experience with its use in diabetics than perhaps any other person, and she said she had had no purpura at all. She did have one death.

For the past nine years we have been testing our pregnant diabetics for blood gonadotropin and, when we find it elevated, we use stilbestrol, up to 150 grams daily.

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This always scares me, because it is so much more than used in any other type of case—at least in my experience. Yet we have had no trouble. The hemoglobin has not dropped in any case that I recall. It seems to be of some real use, particularly if we remember Dr. White's series, in decreasing the fetal mortality rate. In any such problem the danger of the treatment must always be weighed against the danger of the disease. With a fetal mortality rate of from forty to fifty per cent in diabetics you have a real saving of life if you can change that to ten or fifteen per cent, which Dr. White has finally done.

It seems to me this is sufficiently established to be worthy of further trial by other people. If the danger of death to the mother is not more than one in five hundred, which it seems to be, then I think it is worth trying, provided we pay attention to such reports as Dr. Ridgway's in picking up the early toxic signs and in treating the condition properly at the time.

The United States Civil Service Commission

Has announced a Medical Officer examination for filling rotating intern psychiatric resident, and surgical resident positions in St. Elizabeths Hospital, Washington, D. C. The salaries for rotating interns are $2,200 for the first year and $2,400 for the second year; the salaries for psychiatric resident range from $2,400 to $4,100 a year; and for surgical resident, from $3,400 to $4,150.

To qualify, applicants for the rotating intern positions must be third- or fourth-year students in an approved medical school. Applicants for psychiatric resident and surgical resident positions must be graduates of a medical school with the degree of doctor of medicine, and must have completed a full year in an approved rotating internship. In addition to the above requirements, applicants for appointment as surgical resident must have completed three full years as residents-in-training in surgery in an approved residency. No written test is required for this examination. The maximum age limit of 35 years is waived for persons entitled to veteran preference.

Further information and application forms may be obtained at most first- and second-class post offices, from civil service regional offices, or from the U. S. Civil Service Commission, Washington 25, D. C. Applications will be accepted by the Commission's Washington office until further notice.

Living Conditions and Rehabilitation

Living conditions in cities handicap patients recovering from serious illnesses, points out an editorial in the May 21st *Journal of the American Medical Association.*

Convalescents used to be stimulated by the daily needs of life on the farm and in the home, and their relationship with the doctor was sufficiently personal to help in the rehabilitation process, the editorial says.

Nowadays, however, and especially in cities, living conditions do not aid rehabilitation, and the doctor who has served the patient well during his recovery in the hospital can do little or nothing for him during the rehabilitative phase.

Many people live almost monastically in apartments like cubicles. From the hospital an inmate returns to his cubicle, where materials are delivered to the service door by the smoothly flowing "circulatory system" of the metropolis, and activities for recreation tend to be passive ones, such as listening to the radio, reading, and watching television, plays, or sports.

A prolonged convalescence imposes added strains upon a family living in cramped quarters. Modern living conditions in cities make special rehabilitation programs an important part of medical care, and emphasize the role of physical medicine and occupational therapy in the rehabilitation of patients.

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INFLUENCE OF STILBAMIDINE IN MULTIPLE MYELOMA WITH PRE-EXISTING RENAL INVOLVEMENT—CASE REPORTS*

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INTRODUCTION

In 1946 Snapper[1] first presented a series of cases of multiple myeloma in which he had tried two drugs of the di-amidino-stilbene group, stilbamidine and pentamidine. These drugs, particularly stilbamidine, had been used with complete success in the treatment of kala-azar, and the occurrence of hyperglobulinemia in this disease and in multiple myeloma led to their clinical trial in the latter. Results so far have indicated that these agents are useful in relieving the severe pain of multiple myeloma, and in some cases have markedly reduced the invalidism.

Arai and Snapper[2] reported fourteen cases in which particular attention was paid to the influence of stilbamidine on the renal, hepatic, and hematopoietic systems. Of these, three cases showed an increase in the blood non-protein-nitrogen or blood creatinine, with no demonstrable specific lesions at autopsy which might be attributed to the stilbamidine. Two other cases with pre-existing renal involvement revealed only the typical changes of multiple myeloma at autopsy, although the course of their renal disease appeared to have been aggravated by the therapy. In none of the five cases autopsied were hepatic lesions found.

The use of the di-amidino-stilbenes, particularly 4:4′ diamidino-stilbene or stilbamidine, in the therapy of kala-azar has been known since 1939.[1] Most of the experience with this drug was confined to the Middle East and India. Oastler and Fidler[3] reported toxic effects in animals, with widespread damage to liver, kidneys, brain and other internal organs. Sen Gupta[4] criticized this work, asserting that the changes recorded were the result of the use of autoclaved solutions of stilbamidine, with chemical changes causing the toxicity. However, experience with humans indicates that certain toxic effects may be expected. Some of these appear immediately upon injection, such as numbness and tingling of the extremities, or a feeling of formication, and occasionally there is a drop in blood pressure which is transitory and not of great significance. A delayed effect has been noted by most workers two to five months after completion of treatment, consisting of a trigeminal neuropathy with numbness, formication, heaviness and itching.[2] There is dissociated anesthesia, with loss of the sensation of light touch but not of pain, pressure or temperature. There is slow recovery from all these effects except the dissociated anesthesia, which may be permanent.

Seager and Castelnuovo[5] found that the maximum dose of stilbamidine tolerated by mice was 50 milligrams per kilo intraperitoneally, and by rabbits was 20 milligrams per kilo intravenously. The chief toxic effects they observed were in the liver and kidneys. In the liver the changes resembled the precirrhotic changes seen in various kinds of poisonings. Most of the damage was in the periphery of the lobules, with the amount of fibrosis apparently proportional to the survival time of the animal. In the kidney cloudy swelling was seen in the tubules, sometimes with involvement of the glomeruli. Congestion and enlargement of intertubular vessels were observed, while hyaline and granular casts have been found, especially in the lower part of the medulla. Nevertheless, there were distinct species differences, with the mice more resistant to liver damage and the rabbits to kidney damage. It is not clear whether the solutions of stilbamidine used were fresh.

The effect of stilbamidine in human beings has not been completely studied. In this country Snapper and associates,[6,7,1] in using the drug for the treatment of multiple myeloma, have made intensive studies of the deposition of stilbamidine in the myeloma cells of the bone marrow and in myelomatous tissue. They found basophilic inclusion

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bodies in the cytoplasm of myeloma cells in patients treated with stilbamidine, and an appreciable concentration of stilbamidine in myelomatous tissue. The inclusion bodies were demonstrated to contain ribose nucleic acid, and on the basis of their work they advanced an hypothesis for the action of stilbamidine in multiple myeloma. They proposed that the stilbamidine combines with the ribose nucleic acid of the myeloma cell to render it inert, and thus prevent the formation of the abnormal proteins found in multiple myeloma. They suggested that the myeloma cell itself is the principal source of the abnormal globulins, such as Bence-Jones protein, and that stilbamidine has an affinity for the abnormal nucleoproteins involved in their synthesis, but not for the nucleoproteins of normal cells. This is of particular interest, since it is known that actively regenerating cells, such as are found in cancer tissue undergoing active protein synthesis, contain large amounts of basophilic nucleoproteins. This is true of the myeloma cell, but the particular aberration producing the Bence-Jones protein may require a specific nucleoprotein which is chemically susceptible to inactivation by stilbamidine.

While the subject of multiple myeloma has been extensively studied, reviews of the literature from time to time indicate that there are still some controversial areas. Excellent reviews of the subject are available, and it is not our purpose to duplicate them here.[8,9,10,11] Of particular interest are the variations reported in blood calcium and phosphorus levels and their relation to function of the parathyroids. Barr and Bulger[12] reported a case with hypercalcemia, normal blood phosphorus, and hyperfunction of the parathyroid glands. Snapper[9] states that this is rare, with normal parathyroids the rule even with hypercalcemia, while hypercalcemia itself is unusual. The nature of the abnormal proteins found in the blood has not been definitely elaborated, probably because of the wide fluctuations in quantitative determinations of the globulin fractions in different cases of multiple myeloma. The chemical nature of Bence-Jones protein has not been elucidated, although two observers independently have found Bence-Jones protein in the bone marrow[13,14] and there is little question as to its endogenous origin.

Since the number of reported cases of multiple myeloma with pre-existing renal involvement treated with stilbamidine is very small, we are presenting two cases treated at the McGuire Veterans Administration Hospital in the terminal stages of the disease, with autopsy findings, in order to add to the material available for future final evaluation of stilbamidine therapy in multiple myeloma.

Case 1. (H. D., Register No. 8069): The patient was a 59-year old white male whose symptoms began in September, 1946, with soreness of the left shoulder made worse by motion. This was soon followed by pain in the chest made worse by coughing. Treatment for pleurisy did not relieve the pain. It became worse in the thoracic spine and the lower ribs, with right hip pain appearing by the end of May, 1947. There had been no weight loss or other systemic complaints. The patient continued to work until April, 1947. During the Spring of 1947 his doctor noted proteinuria. Urography revealed bony abnormality which was interpreted as possible hyperparathyroidism. Further workup showed a high blood protein with hyperglobulinemia, Bence-Jones proteinuria, and skull X-ray findings consistent with multiple myeloma. Slight shortening of stature was noted. He was admitted to the Veterans Administration Hospital in Huntington, West Virginia, on June 3, 1947, and transferred to McGuire Veterans Administration Hospital, Richmond, Virginia, on July 2, 1947.

At Huntington physical examination revealed a well-developed, well-nourished elderly white male lying quietly in bed, but experiencing a good deal of pain in the lower thoracic spine on movement. Pressure over the lower ribs bilaterally caused pain radiating around to the back. BP was 118/80. The liver was palpable two finger-breadths below the right costal margin on deep inspiration. There was slight bilateral costo-vertebral angle tenderness, with tenderness on percussion of the vertebrae from D-9 down to L-1. There was also tenderness over the sternum. X-rays showed sharply-demarcated, punched-out areas varying from three to twelve millimeters in the skull, ribs, lumbar vertebrae, pelvis and femurs. Pathological fractures of the left and right third ribs and left fifth rib were noted, with collapse of vertebrae D-7 and D-8 and cortical thinning of D-9 and D-10. Beginning collapse of L-1 and L-5 was seen. Chest X-ray showed increased broncho-vascular markings with fibrosis and circumscribed areas of parenchymal infiltrations. The peripheral blood showed a slight leucocytosis. Blood calcium was 15.4 mgm. per cent,

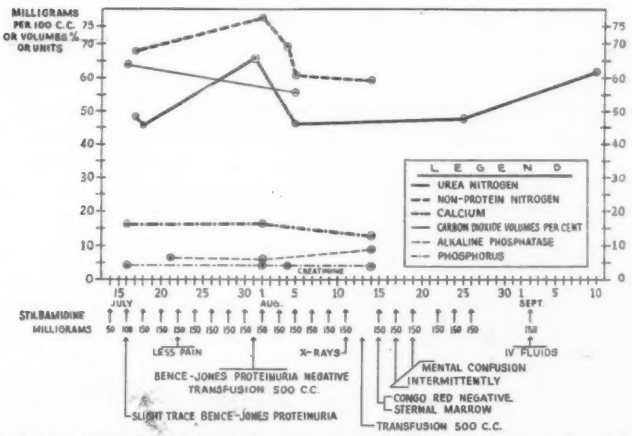

Fig. 1.—Graph showing blood chemistries, and other tests and treatments while under stilbamidine therapy.

inorganic phosphorus 6.1, non-protein nitrogen 38.7, creatinine 1.0. The total blood protein was elevated to 9.25 grams per cent, with albumin of 2.67 grams and globulin of 6.58 grams. Urine was negative for Bence-Jones protein, although repeated urinalyses showed constant 4 plus albuminuria with many hyaline and finely granular casts and maximum concentration to specific gravity of 1.017. Sternal marrow examination was pathognomonic for multiple myeloma, plasma cell type. The patient was completely bedridden because of pain in his chest and back. He developed anorexia and constipation, with progressive weight loss.

When the patient arrived at McGuire V. A. Hospital he was poorly nourished and showed evidence of chronic weight loss. There was general pallor of the mucous membranes. The chest was emphysematous in type, with a few fine rales at the lung bases. The tendon reflexes of the upper extremities were hyperactive and there was a coarse fascicular twitching in the arms and hands.

The laboratory findings are summarized graphically, together with pertinent clinical events which may have influenced them. Figure 1 is a presentation of the blood chemistry findings, while Figure 2 presents the peripheral blood picture. The urinary findings showed little change, with persistent 4 plus albuminuria and gradual lowering of the specific gravity. PSP dye excretion was markedly diminished, totaling less than 15% in two hours on July 15, 1947. On August 21 quantitative urine albumin determination showed 2.5 grams per liter per 24 hours, with 3.2 grams per liter on September 12.

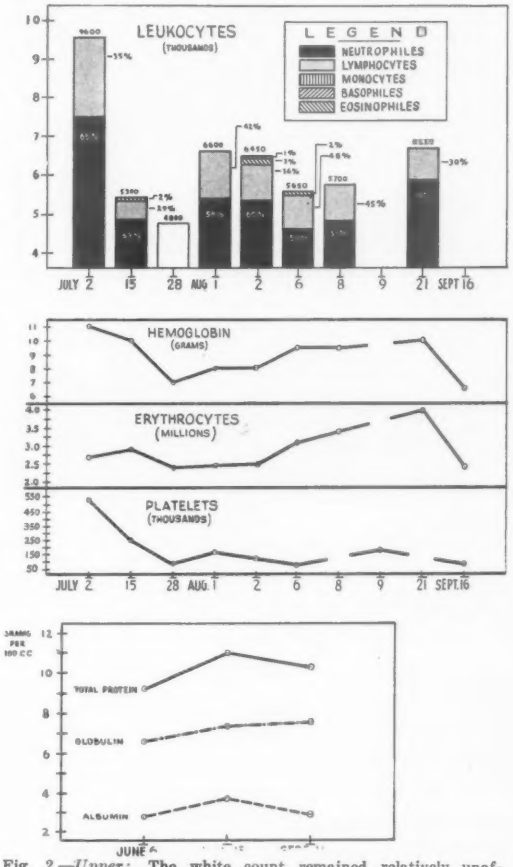

Fig. 2.—*Upper:* The white count remained relatively unaffected during the course of treatment.
Middle: Progressive myelophthisis affecting the erythropoietic and platelet elements occurring despite transfusions and stilbamidine therapy. The platelet count at times fell below the critical level.
Lower: Hyperglobulinemia with elevated total protein and reversal of the A-G ratio is demonstrated.

Bence-Jones proteinuria was found on only one occasion, despite numerous analyses by special technique. Sternal marrow examination on July 2 revealed 80% of the myeloid cells to be of the plasma cell type (Plate I). A second sternal marrow examination on August 15, after the patient had received fifteen injections of stilbamidine, totaling 2,100 mgm., revealed no essential change. Repeated studies of the peripheral blood revealed an occasional plasma cell. Basophilic inclusion bodies, as described by Snapper, were not seen in the sternal marrow examination of August 15.

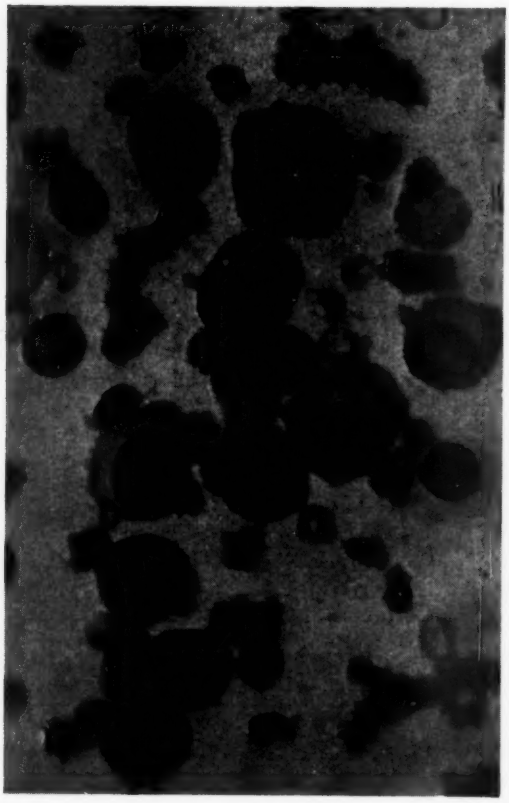

Plate I.—Photomicrograph of sternal marrow in first case. Typical myeloma cells of plasma cell type are seen. Wright's stain.

X-rays taken July 8 showed some progression of the bony lesions in the one month interval since the last series. The right eighth rib had a pathological fracture. Areas of decreased density were noted in the scapulae and clavicles. The spine likewise showed numerous rounded punched-out areas of decreased density in most of the vertebrae, with flattening and anterior wedging in D-4 and D-5, D-8 and D-9, D-12 and L-1. Osteoporosis was present in all bones examined. The renal shadows appeared normal. The lungs appeared normal. Re-examination of the skull, ribs, spine and pelvis including upper one-third of the femurs on August 12 showed evidence of definite increase in size and number of the osteolytic defects (Plate II). The patient had completed his first course of stilbamidine at the time of the repeat X-rays, and had received fifteen injections totaling 2,100 mgm.

During therapy, the clinical course was at first encouraging. After four injections he noted a definite lessening of the pain in his back. He continued to show symptomatic improvement with lessening of pain everywhere but in the sternum. He was able to move about in bed, and on several occasions sat

Plate II.—*Upper X-Ray:* Before treatment.
Lower X-Ray: After treatment. Note the increase in size and number of osteolytic defects.

up or kneeled spontaneously. His general condition, however, showed steady deterioration. Anemia and azotemia became more marked, responding at first to supportive measures, but with less response progressively. Stilbamidine therapy was continued until the patient had received a total of twenty-two injections, or 3,150 mgm., by September 2. He had become increasingly drowsy and lethargic during the last two weeks of treatment, and at times appeared confused mentally. His nutritional intake became very inadequate, and attempts to render further supportive treatment intravenously were unsuccessful because of venous thrombosis subsequent to the stilbamidine therapy. On September 16 he had a rectal hemorrhage of about 200 cc. of bright red blood. Rectal examination disclosed a fecal impaction with tarry stool and tarry liquid feces behind the impaction. Platelet count at that time was 80,000. Drowsiness progressed to stupor, cachexia was marked, and the patient died in respiratory collapse on September 19, 1947.

Autopsy Findings: A complete post-mortem examination was performed 15½ hours after death. The body showed marked emaciation and cachexia. The significant gross and microscopic findings were: Heart—weight 250 grams. The myocardium was reddish-brown and firm. The coronary arteries were patent. Microscopically, no remarkable changes were noted.

Lungs—The right weighed 475 grams; the left 550 grams. Focal areas of consolidation were noted in both lungs. Microscopically the lungs showed broncho-pneumonia.

Liver—weight 1150 grams. The capsule was smooth. Microscopically fatty metamorphosis of the liver cords and a few areas of lymphocytic infiltration in the interlobular connective tissue were present.

Spleen—weight 50 grams. The capsule was smooth. The cut surface was purplish-red, and the pulp sank below the level of the capsule. The Malpighian corpuscles were indistinct. The most striking change microscopically was the marked deposition of hemosiderin found both intra and extracellularly.

Kidneys—The right weighed 100 grams; the left 150 grams. The capsules stripped with ease revealing finely granular surfaces. Microscopically many of the glomerular tufts showed hyalinization of the capillaries. Many convoluted and collecting tubules were occluded with homogeneous hyaline-appearing casts that showed numerous linear cracks. In the neighborhood of these protein casts were giant cells of the foreign body type. There was round cell infiltration of the interstitial tissue. A few calcific deposits were seen in the medulla.

Skeletal System—pathologic fractures of the 2nd and 7th ribs on both sides were noted. On the pleural surfaces of the right 3rd and 4th ribs just lateral to the costovertebral junction a bulging soft, fluctuant tumor mass was seen. This tumor mass was directly connected with the bone. Over the manubrium sterni there was another area where the bone was replaced by soft, necrotic-appearing tumor tissue. Microscopically the bone sections showed the characteristic findings of multiple myeloma.

Skull—no fractures were found. However, numerous small circumscribed areas, the largest measuring 1 cm. in diameter, were seen. These were depressed below the bone surface, soft and hemorrhagic. Microscopically these showed typical multiple myeloma.

The adrenals, prostate, brain, pituitary, thyroid, parathyroids, and pancreas did not reveal significant changes.

Case 2. (M. R., Register No. 7728): The patient was a 57-year old Negro male admitted to the hospital originally on June 16, 1947, for treatment for pulmonary tuberculosis. He gave a history of pain in his chest and cough productive of bloody sputum for two months prior to admission. The week before admission he had been treated with streptomycin by his local physician. A chest X-ray revealed tuberculosis, and the patient was hospitalized. The only other symptoms of note were pain in the left hip on motion of that joint since March, 1947; transient pain in both upper abdominal quadrants for two weeks; nocturia five to six times, with daytime frequency; and "chest trouble" since serving in France in World War I.

Physical examination revealed a well-developed, fairly well-nourished, middle-aged Negro male, who was slow in his responses and appeared to be below normal in intelligence. The history was elicited with great difficulty. Medium moist rales were scattered throughout the right upper lung field. There was some tenderness on pressure in both upper quadrants. Pain was elicited on motion of the

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left hip in abduction and flexion. There was no local swelling or increased heat.

On the day of admission to the hospital posteroanterior and apical views of the chest by roentgenogram showed a fibrotic infiltration in the upper half of the left lung field with considerable diminution in size of the left hemithorax. A few nodules were seen in this area. On the right there was a fibrotic infiltration overlying the second rib, with mild fibrosis and emphysema in the remaining lung fields. Several old healed fractured ribs were seen. The roentgen appearance was consistent with tuberculosis of neoplasm. The patient's serum was found to be anticomplementary because of hyperglobulinemia. X-rays were then taken on June 23 of the bony pelvis, spine, skull, long bones, ribs and both hands. These revealed widespread oval and punched-out areas of decreased density in all bones examined except the spine and hands, where arthritic changes of the hypertrophic type were evident. Sternal marrow puncture was then performed, and the diagnosis of multiple myeloma was established on June 26 with the finding of 40% myeloma cells of the plasma cell type in the bone marrow smear. Peripheral blood smears on June 27 showed 3% plasma cells, but numerous subsequent studies were negative except for a rare plasma cell seen on August 14. The disease in this patient remained confined to the bone marrow.

TABLE I

Date	*Hgb.* (gms.)	*Rbc.* (millions)	*Wbc.*	*Sed. Rate*	*Hematocrit*	*Platelets*
6/16	10.5	2.95	6950	mm/hr.		
6/21	10.0	3.65	6000			
6/22	10.5	3.31	5300			
6/24	8.0	2.80	7350	26	29	
6/27	11.0	3.49				
6/28	11.5	4.38				
7/1	14.5	4.63	Transfusions			
7/2	11.5	3.98				
7/3	11.0	4.20				
7/7						590,000
7/9	10.5	3.65				
7/10	9.5	3.28				
7/23	9.0	3.35				
8/1	9.0	2.95				
8/2		3.84	15,800			200,000
8/7	8.0	3.00	11,050	5		240,000
8/9	10.0	4.08	7,100			110,000
8/12	10.5	3.60	9,200			130,000
8/14	11.0	3.48	8,300			120,000
8/16	8.5	2.91	8,700			
8/19	10.5	3.60	6,900			110,000
8/21	9.0	3.07	8,500			150,000
8/23	9.5	3.00	7,550			150,000
8/26	8.5	2.75	9,850			150,000
9/1	9.5	3.20	9,100			170,000
9/13			7,700			

Case 2. **Progressive anemia developed without demonstrable influence on the platelet or white blood cell count.**

Laboratory studies are presented in tabular form where possible. Table I lists the blood counts, and reveals a continuous progressive anemia which showed only temporary response to transfusions. The white cell count and platelet count did not show any abnormality throughout the patient's illness. The blood chemistries are presented in Table II.

TABLE IIA

Date	*Formol gel*	*T.P.*	*Alb.*	*Glob.*	*Chlor.*	*BSP*	*Ict. Ind.*	*Acid Phos.*	*Vd B.*
6/17		9	3.7	5.3					
6/18							6		neg.
6/19						4% retention			
6/24	positive	8.7	3.9	4.6	462				
6/28								neg	

TABLE IIB

Date	*Cal.*	*Phos.*	*Alk. phos.*	*BUN*	*NPN*	*Creat.*
8/7	16.7	3.9	11		65	
8/12					67	
8/15					76	
8/19					85	
8/22				40	56	
8/26				62		
9/13				37		2.5

Tables IIA and IIB. Blood chemistry tests performed in *Case 2.* Note that the degree of azotemia had wide fluctuations, apparently unrelated to stilbamidine therapy.

More determinations were not done because of the technical difficulty in obtaining blood in sufficient quantities as a sequel to the venous thrombosis which occurred locally following the injection of stilbamidine. It is interesting to note that on two out of three occasions the sedimentation rate was normal despite the high globulin content of the serum and the presence of active tuberculosis.

Bacteriological studies of sputa and gastric washings were negative for acid-fast bacilli, although a bedside chest film on September 12 suggested extension of the changes in the right upper lobe. The urine showed a persistent trace of albumin with a low fixed specific gravity which reached a maximum of 1.007 during a concentration test on August 8,

pyuria ranging from four to thirty cells per high power field, intermittent hyaline casts, and varying amounts of renal epithelial cells. No Bence-Jones protein could be discovered, however. Quantitative measurement of the urine albumin excretion in 24 hours was 0.92 grams on August 13, at a time when the azotemia was increasing. Further X-ray studies were performed. A bedside chest film on September 12 revealed marked collapse of the bodies of the third, ninth and eleventh thoracic and first lumbar vertebrae. Other bony changes were probably more marked than on previous examination. X-ray of the cervical spine on August 25 showed no evidence of pathological fracture.

Incidental laboratory studies showed a negative Wassermann, negative Frei test, and cerebrospinal fluid total protein of 59 mgm. per cent with all other spinal fluid findings normal. Biopsy of rectal mucosa after an episode of rectal bleeding was negative, while biopsy of a mass excised from the left prepatellar region revealed a fibroma. Barium enema following transient severe abdominal pain was negative.

The patient's course in the hospital was stormy. He had transfusion reactions of a febrile nature, and thereafter had febrile episodes intermittently. He went progressively downhill, and became increasingly more lethargic. His pain increased, so that he was completely immobile in bed and could be turned only with great difficulty. Most of the pain appeared to be in the left hip and pelvis. Stilbamidine therapy was begun on August 7 and given every other day intravenously until a total of 1.2 grams in nine injections had been given. Toward the end of the treatment period there was a sharp rise in the blood non-protein-nitrogen. The patient was drowsy, but on persistent questioning stated that there had been no change in the pain. At the same time, administration of the drug had become very difficult because of local venous thromboses. For these reasons, and because the patient's condition made further evaluation of the drug doubtful in this case, the therapy was discontinued. At the end of August the patient developed a transitory myositis on the left side of his neck. Emaciation had become marked with a decubitus ulcer over the left gluteal region. The patient became semicomatose with gradual deterioration until death on September 24, 1947.

Autopsy Findings: A complete post-mortem examination was performed 21 hours after death. The body showed marked emaciation. The significant gross and microscopic findings were: Heart—weight 260 grams. Myocardium reddish-brown and flabby. Coronary arteries tortuous but patent. Microscopically no significant changes were seen.

Lungs—the right weighed 780 grams, the left 650 grams. Both lungs showed evidence of extensive tuberculosis with cavitation. Microscopically the lungs, parietal pleura and peribronchial lymph nodes were tuberculous.

Liver—weight 1330 grams. The capsule was smooth. The left lobe was narrow and elongated. A thick band of fibrous tissue separated the tip from the body of the left lobe. Microscopically sections from the right lobe showed round cell infiltration in the interlobular connective tissue. Focal areas of necrosis were seen surrounded by leukocytes. The liver cords showed marked bile pigment in the cytoplasm. Sections from the tip of the left lobe showed complete destruction of the lobular structures. The liver cells were necrotic and only a fine reticulum was seen in which could be seen pigment-laden macrophages, lymphocytes and degenerated liver cells. Groups of biliary ducts were identified with considerable round cell infiltration around them. Numerous bands of scar tissue and thick-walled blood vessels were seen.

Spleen—weight 100 grams. The capsule was wrinkled. The cut surface was dark red and sank below the level of the capsule. The Malpighian corpuscles were indistinct. Microscopically, there was a heavy deposition of hemosiderin both intra and extracellularly.

Kidneys—the right weighed 120 grams, the left 140 grams. The capsules stripped with slight difficulty, revealing finely granular surfaces. Microscopically, a few of the glomeruli showed fibrosis. Focal areas of lymphocytic infiltration were seen. Many of the collecting tubules were filled with hyaline-appearing casts that showed linear cracks. Mild sclerosis of the blood vessels with hyalinization of the walls was seen.

Skeletal System—pathologic fractures of 3rd to 6th ribs right, and 3rd to 5th ribs left were noted. Microscopically, the sections showed the typical findings of multiple myeloma.

Skull—many discrete, soft hemorrhagic areas

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were noted in the calvarium and at the base of the skull, particularly about the posterior and anterior clinoid processes. Microscopically, the sections disclosed typical multiple myeloma.

The remaining organs were essentially normal.

COMMENT

Both of these cases presented the typical picture of advanced multiple myeloma. Neither case showed evidence of amyloidosis despite the extensiveness of the disease and, in the second case, its association with tuberculosis. Hypercalcemia was present in both, with calcific deposits microscopically in the kidneys in one. The parathyroids were apparently normal. Hyperglobulinemia was present in both, although Bence-Jones protein was found in only one. Typical bone lesions were present with marked invalidism, and the bone marrow smears were pathognomonic.

Because of the advanced stage of the disease, and the marked renal involvement in both cases, therapy with stilbamidine was begun cautiously with administration of the drug every other day intravenously. Marked symptomatic response was observed in our first case after the fourth injection, although all X-ray and laboratory studies showed a continuous progression of the disease. Pain was materially reduced, and movement in bed became possible. The effect on the kidneys was not sufficiently marked to attribute any of it to the stilbamidine. At first there seemed to be an increase in the uremia, but transfusions and intravenous fluids reversed this tendency, and there was no further significant rise despite the continuance of treatment until a total of 3150 milligrams had been given. Although there was a progressive downhill course with lethargy and drowsiness, death actually came because of bone marrow phthisis and intractable progressive anemia, due to widespread replacement of the normal marrow with myeloma tissue, and terminal thrombocytopenia.

In our second case treatment was discontinued after nine injections, or after a total of 1200 milligrams of stilbamidine had been given. The patient clinically was semi-stuporous part of the time, and showed no apparent clinical or symptomatic benefit from the drug. He remained motionless in bed because of pain, and showed no evidence of palliation. There was a steady increase in the extent of his uremia with an uremic death, associated with progression of the pulmonary tuberculosis.

In both cases transitory paresthesias were noted during administration of the drug, but no other ill-effect. A clinically important tendency of the drug to produce venous thrombosis locally at the site of injection was noted, the thrombosis usually requiring one to two days to develop. This interfered with the supportive treatment of the patient, and it is recommended that in further cases the drug be used in greater dilution in normal saline. Freshly prepared non-autoclaved solutions of stilbamidine were used in both cases for each injection.

At autopsy the gross findings were essentially those to be expected. The microscopic findings differed somewhat from the gross, particularly with reference to changes observed in the liver. In the first case, a chronic inflammatory reaction of the interlobular connective tissue, with fatty changes in the liver cord cells, was observed. These changes were non-specific, and could have been due to the marked cachexia, rather than to the stilbamidine. In the second case a varied picture was seen in the liver, ranging from focal areas of mild inflammation and discrete necrosis to complete dissolution and destruction of liver tissue. Here again interpretation is difficult. The inflammatory reaction might have been due to cachexia or the stilbamidine. The area of greatest destruction was found distal to the congenital band or post-traumatic scar. The patient was not alert enough on arrival in the hospital to give an adequate past history. Only further experience with stilbamidine will determine whether the lesions in the left lobe were due to the drug. No jaundice was ever observed clinically in our patients, and the microscopic lesions were not considered to be of fatal extent.

A significant finding was the lack of any lesion in the kidneys attributable to stilbamidine. The findings of advanced "myeloma kidneys" were present.

In our sternal bone marrow studies we were unable to demonstrate the inclusion bodies described by Snapper, although clinically satisfactory palliation was achieved in one case.

Neither of our patients lived long enough after treatment to develop trigeminal neuropathy.

There were no extra-medullary metastases in either case.

SUMMARY

1. Two cases of multiple myeloma with marked renal involvement, treated with stilbamidine, are presented.

2. No lesions were found in the kidneys attributable to the drug. Non-specific lesions were present in the liver. Further experience with stilbamidine is necessary to evaluate these properly.

3. Local venous thrombosis at site of injection was encountered frequently. Much greater dilution of the drug at time of injection is indicated.

4. Palliation of pain was achieved in one case. There was no apparent effect on the course of the disease in either case.

We wish to express our appreciation to Merck & Company, Inc., who supplied the stilbamidine used in this study.

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Persons With High Blood Pressure Lack Self-Assertion.

A significant association between high blood pressure and lack of assertiveness is pointed out by five St. Louis doctors.

The doctors—Gregory C. Gressel, M.D., Frank O. Shobe, M.D., George Saslow, M.D., Henry A. Schroeder, M.D., and Philip H. DuBois, Ph.D., of the Departments of Neuropsychiatry, Psychology, and Internal Medicine of Washington University—made a study of personality patterns in 50 patients with high blood pressure as part of a comprehensive investigation of the disease.

Reporting in the *Journal of the American Medical Association*, the doctors say that a comparison of personality patterns of these patients and of those of 49 patients with personality disorders and 44 patients with chronic illness of physical origin shows statistically significant degrees of association between high blood pressure and "subnormal assertiveness" and "obsessive-compulsive behavior."

Traits of being excessively submissive, ingratiating, and apologetic, and of greatly repressed feelings of hostility were found in 34 of these patients with high blood pressure, in 20 of the patients with personality disorders, and in only six of the patients with chronic illness of physical origin. Handicapping traits of meticulousness and repetition of actions were found in 37 patients in the first group, 14 in the second group, and six in the third group.

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THE ROLE OF THE LIVER IN BLOOD COAGULATION*

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The clotting process is, as Tocantins has pointed out again in a recent article, only one of several factors that produce hemostasis. Other factors to be considered are the location of the vessel, the tension of the surrounding tissue and the size and anatomical structure of the vessel. The condition of the vascular lining and the rate of blood flow also are of the greatest importance. Nevertheless, the clotting mechanism proper and the agents involved in it have always attracted special interest and still do, as the six hundred odd articles indicate that are listed on this subject matter in the *Annual Reviews of Physiology* of 1946 and 1948.

The purpose of this presentation is to review briefly the role of the liver in blood coagulation. Limitation of time makes it necessary to confine these remarks to facts that seem well established, omitting many interesting but controversial aspects of the matter. I shall take up one after another the various substances involved in the clotting process as they are related to the liver, discuss briefly their nature, function and significance, and thus highlight the central many-sided and indispensable role which the liver occupies in blood coagulation.

The present prevalent concept of clotting divides this process in four consecutive phases: the first step is preliminary and represents what can be called the trigger mechanism. It consists in massing and clumping of platelets, resulting in their disintegration and the release of thromboplastic substances. At the same time, a reduction of antithromboplastin, normally present in the plasma, occurs. Under physiological conditions, only minimal amounts of thromboplastic substances are liberated from platelets and are immediately inactivated by antithromboplastin, also called anticephalin. It is chiefly this absence of free thromboplastin which maintains the blood fluid in our vessels. During the second phase of clotting—the conversion phase—prothrombin, by the action of thromboplastin, is transformed into thrombin. The amount of thrombin formed is proportional to the amount of prothrombin available.

*Read at the Spring meeting of the Medical Society of the Valley of Virginia in Clifton Forge, May 28, 1948.

Prothrombin is formed in the liver and will be discussed in greater detail. The presence of ionized calcium is necessary in this step but the mechanism of its action is still unsettled. In the third phase, thrombin acts on the sol fibrinogen to form the gel fibrin. Fibrinogen is another substance formed in the liver and we shall come back to it. Finally, in the fourth phase, the formed clot is eventually dissolved by a normally present substance, fibrinolysin.

Prothrombin is an euglobulin containing about 4.3 per cent carbohydrate and an active alpha amino group. It appears fairly well established that there are two variations of prothrombin: a labile type, prothrombin A, which disappears from stored blood; and a prothrombin B which disappears following administration of dicoumarol. The practical application of this is that banked blood is perfectly acceptable for the treatment of dicoumarol over-dosage. As already said, prothrombin is formed in the liver. Adequate supply of vitamin K and good hepatic function are prerequisites for its production. Prothrombin is not measured quantitatively in the blood but by its activity, usually by the one stage method of Quick. Its principle consists in determining the clotting time of plasma with thromboplastin and calcium ions present in fixed and optimal amounts. There exists considerable disagreement on whether the clotting time under such conditions is actually and exclusively dependent on the concentration of prothrombin but the test meets all practical requirements. It must be kept in mind that the relation between plasma clotting time and prothrombin concentration is not expressed by a straight line but by a hyperbolic curve. Thus, if we call a clotting time of 12 seconds normal and 100 per cent, a clotting time of 15 seconds would mean a reduction of prothrombin to 50 per cent. A clotting time of 25 seconds would represent a prothrombin level of 20 per cent of normal, one of 38 seconds, 10 per cent of normal. Increased prothrombin activity, or, in other words, a shortened clotting time, may be found in late pregnancy and following the administration of digitalis, methylxanthines and adrenalin. Since digitalis also seems to release thromboplastin from the platelets, its

use in cardiac failure complicating coronary thrombosis involves definite hazards. Clinically more important, however, is reduction of prothrombin activity, as expressed by prolonged plasma clot-long time. Such reduction takes place due to increased utilization of prothrombin as in trauma and chronic illness. It is present in idiopathic hypoprothrombinemia and is brought on by a number of drugs as dicoumarol, salicylates, barbiturates and various hepatotoxic agents like chloroform and carbon tetrachloride. It follows lack of vitamin K and is found in liver insufficiency. Vitamin K is essential for the production of prothrombin. It is not a clotting factor itself but an indispensable link in liver metabolism which leads to prothrombin formation. How it acts in this respect is unknown. Radioactive vitamin K has been seen to diffuse rapidly without being retained in the liver. The various forms of vitamin K all contain an active naphthoquinone ring. It is interesting to note that the much more effective synthetic compounds are chemically much simpler substances than the natural vitamins. Vitamin K deficiency due to inadequate intake is practically non-existant since supply is abundant and synthesis by the intestinal flora continuous. It might be mentioned in this connection that sulfa drugs do not interfere with this synthesis. An exception is the biological deficit in the new born due to the absence of intestinal bacteria, and the inability of the infant to store the vitamin. The advantage of providing an extra supply to the infant by vitamin K administration to the pregnant mother is recognized despite certain statistical evidence that intracranial and retinal hemorrhage in the newborn is not significantly reduced as compared with untreated controls. Failure of absorption is seen in bile duct obstruction uncomplicated by hepatic damage. It is due to the absence of bile salts in the intestinal tract. It is also found in sprue, regional ileitis and chronic diarrhea. It may follow excessive use of mineral oil with resulting loss of the fat soluble vitamin. Failure of absorption can be corrected by administration of vitamin K and bile salts. This is not true when associated liver damage is present, as in long standing obstruction of the common duct. As a matter of fact, the lack of response to vitamin K therapy in such cases has been considered a measure of the liver damage and a liver function test based on this principle has been advocated. It does not seem, however, that this test is more helpful than others in differentiating long-standing jaundice of extrahepatic and hepatocellular origin.

The two chief antagonists of vitamin K are dicoumarol and salicylic acid. Both substances reduce prothrombin activity, and prolongation of the plasma clotting time results from their use. *Vice versa,* over-dosage can be corrected by sufficiently large amounts of vitamin K and blood transfusions. Salicylic acid is an intermediary link in the synthesis and breakdown of dicoumarol. It is about one-twentieth as effective and, while it reduces prothrombin regularly, it rarely depresses its level below 20 per cent. With the widespread indiscriminate and unsupervised use of salicylates, these facts are of significance. The routine administration of vitamin K together with large doses of salicylic acid in acute rheumatic fever has been advocated by some authors. Pregnancy and lactation reduce vitamin sensitivity in the mother but sucklings are sensitive to the milk transmitted dicoumarol and therefore mothers receiving dicoumarol should not be allowed to nurse. Dicoumarol also affects thomboplastin and fibrinogen and thus interferes with the clotting process in different ways.

Thrombin is the active clotting enzyme. It is a glucoprotein containing about 5 per cent carbohydrate. It is derived from prothrombin. It controls the rate of coagulation in phase three. It can coagulate fibrinogen up to 200 times its own weight and certain snake venoms with thrombin characteristics actually can clot fibrinogen up to 60,000 times their own weight. The topical application of thrombin for the control of bleeding is well known and will not be discussed here. Any excess of thrombin is inactivated by antithrombin through the formation of metathrombin. This occurs only after all fibrinogen has been disposed of and serves chiefly to prevent extension of thrombin activity to adjacent tissues. Antithrombin activity is potentiated by heparin and hirudin.

Fibrinogen, the precursor of fibrin, is a globulin containing a lipoid fraction. It, too, is formed in the liver. Its blood level is between 140 and 400 mgs. per cent. Increased amounts of fibrinogen are found in infection. It is reduced in severe liver damage by direct suppression and indirectly by reduced output of prothrombin and consequent lack of thrombin. It is also depressed by dicoumarol,

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indicating a direct action of dicoumarol on the liver aside from its antagonism to vitamin K. The function of fibrinogen in clotting involves phase three where it quantitatively determines the amount of fibrin formed. Even more important is its role in immunity as exemplified by the fibrinous exudate in pneumonia and wound repair.

Heparin has puzzled everybody since Howell and his coworkers extracted it from the liver. Its chemical nature is not fully established but it is known to be a mucoitin sulfuric acid. It is formed in the mast cells of the body, especially those in the liver and the lungs. It was noted in victims of the atomic bomb that the mast cells were increased whenever the patients had shown general bleeding tendency. Contrarywise, by injecting toluidine blue which stains and blocks the granules in the mast cells, heparin production can be reduced. Heparin has a threefold action: it inhibits platelet agglutination and thus prevents liberation of thromboplastin. It retards,, with a plasma cofactor, the conversion of prothrombin to thrombin. It forms, with serum albumin, antithrombin. Its therapeutic use is determined by its advantages and disadvantages as compared with other anticoagulants. It hardly produces any bleeding tendency and does not require laborious daily laboratory tests for the control of dosage. It is non-toxic. On the other side, it is still expensive. It requires multiple daily injections and, if suspended in media which slow down absorption, injections are painful. It is used extensively abroad and seems to gain favor in this country. Besides its use in the treatment of thrombosis, it has also found application in the treatment of toxemia of pregnancy and in the prevention of anaphylactic shock.

How the clotting mechanism is affected, through the liver, by the autonomous nervous system, is well demonstrated by the interesting experiments of De Takats. Sympathetic stimulation increases the tendency to thrombosis while cholinergic stimulation decreases it by the dilatory and constrictive effect on the hepatic veins and the resulting greater and smaller outflow of heparin and prothrombin.

CONCLUSION

The liver is directly and indirectly, in various ways, essentially involved in all phases of the clotting process. The role of the particular substances produced or controlled by the liver reaches beyond the coagulation of blood into the fields of immunology and anaphylaxis. Normal liver function—that is cellular integrity—is essential for maintained balance and is controlled by humoral and nervous factors.

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Sodium-Free Salt Substitute Again Available.

Manufacture and marketing of Neocurtasal has been resumed by Winthrop-Stearns, Inc., according to announcement by Dr. Theodore G. Klumpp, president.

On February 18, the Federal Food and Drug Administration suggested recall of salt substitutes containing lithium chloride, following reports of untoward human reactions which may have been due to lithium chloride. Two weeks later FDA recommended to manufacturers that distribution be discontinued of all salt substitutes even though some of them like Neocurtasal, contained no lithium chloride; and it further announced that all such substitutes would henceforth be regarded as new drugs rather than as foods. Under the Food, Drug and Cosmetic Act, a new drug may not be marketed unless and until a new drug application becomes effective with respect to such product.

SURGICAL TREATMENT OF ACUTE GALLBLADDER IN THE FIRST 72 HOURS*

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Philadelphia, Pennsylvania.

Your committee has asked me to speak on the topic, "Surgical Treatment of Acute Gallbladder in the First 72 Hours". I can answer this categorically by saying that I would not operate on an acute gallbladder in the first 72 hours if the symptoms have not subsided. But before we enter into the discussion of this topic, let us consider some specimens of gallbladders removed at operation to determine whether we are all talking about the same subject, because very often, when the specimen is sent to the pathologic laboratory, a report will be returned "subacute or chronic cholecystitis". Clinically, the symptoms and physical signs do not bear out this diagnosis of the pathologist.

We do not operate precipitately on these patients. For example, E. P., age 38, had two attacks of pain sometime prior to admission. She came in acutely ill with high fever and jaundice. She was not operated upon until seven days after admission when all acute symptoms had subsided. An old empyema of the gallbladder and a dilated duct were revealed. On account of the dilatation of the common duct a cholecystectomy and choledochostomy were performed. Fig. 1. To illustrate that we

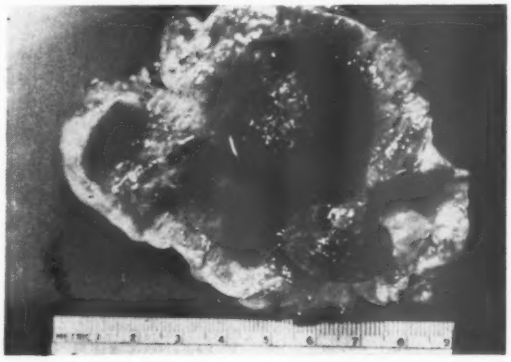

Fig. 1.—E.P. This patient was not operated upon until seven days after admission. An empyema of the gallbladder with gangrene and stones was removed.

are not unmindful of the existence of perforations of the gallbladder, we wish to present the case of J. D., age 40, who was admited to the hospital acutely ill with fever, nausea and vomiting. A diagnosis of perforation of the gallbladder was made. On account of the dangers concomitant with this condition, she was operated upon the day of admission. No perforation was found. Instead, a large gallbladder filled with pus was removed. Fig. 2. Another case of acute empyema of the gallbladder will illustrate a large organ with obstruction of the cystic duct. A cholecystectomy was performed after the acute symptoms abated. Fig. 3.

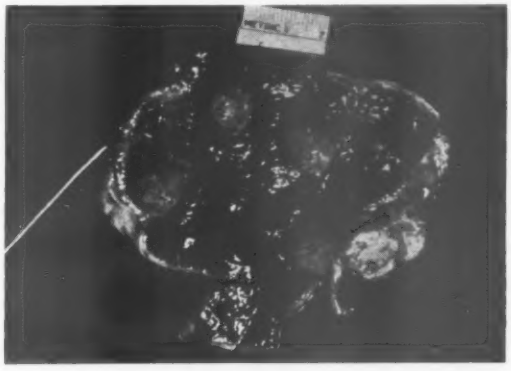

Fig. 2.—J.D. Diagnosis of perforation of the gallbladder was made. Operated upon the day of admission. An unperforated empyema of the gallbladder and cholelithiasis were found at operation.

Fig. 3.—F.S. Gangrene of the gallbladder. Cholecystectomy was performed after subsidence of the symptoms.

Now that we have placed before you the character of the case we describe as an acute gallbladder, the clinical symptoms may not be amiss. The characteristic symptoms on admission are fever and con-

*Read before the Norfolk County Medical Society, December 13, 1948.

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siderable tenderness and rigidity in the right upper quadrant accompanied at times with chills and fever. The physical findings in the right upper quadrant are important. A large pyriform mass outlining the gallbladder is constantly present in these patients. To palpation, this mass is extremely painful. The favorable or unfavorable course of the patient can be followed by palpation of the gallbladder alone. All physicians and surgeons do not agree with this observation, but the diminution in size of an acutely inflamed gallbladder is noticeable by palpation from day to day.

The treatment necessary to attain the diminution of the symptoms is that the patient must be hospitalized and preferably on the surgical service because the internist, unless he has been educated to the manner in which these patients should be treated, does not know how to conduct the preoperative treatment.

It must be evident that acute empyema of the gallbladder is not a medical disease. In some hospitals, the mistake is made of assigning these patients to the medical service, and, in many instances, the internist has been found wanting in the treatment of acute cholecystitis. The internist must recognize the value of this regimen and not feed his patient by mouth, as a result of which perforations and progressive inflammation of the gallbladder are apt to occur. There is practically no danger of perforation if the above regimen is observed. Over 95% of acute gallbladders will subside under proper treatment.

The immediate medical treatment of these patients is absolute abstention of all foods including water. Parenterally, the patient receives about 3000 cc. a day of 5% glucose and amigens. Blood transfusions are given if necessary. Within the first twelve hours, there is a diminution of the symptoms and, after the first 24 hours, there is a very marked reduction of the tenderness, pain and size of the gallbladder. In addition, as a result of this preoperative care, we have not observed a case of liver death in many years.

We do not operate on these patients as an emergency case. On admission, laboratory tests such as the examination of the urine and blood are made, and an estimation of the amount of urea and sugar is determined and a blood sedimentation test may be of value. No one should consider operating upon these cases as an emergency unless the diagnosis of perforation has been made. Furthermore, because most of these patients have passed the age of 40, it is essential to examine the veins of the leg and perform a prophylactic ligation of any patient in whom this is deemed necessary. These operations can be done simultaneously with the abdominal work. This is feasible provided we have capable assistants who can do the leg work while the surgeon and his assistants perform the cholecystectomy A Levine tube is always passed soon after adm nis sion to the hospital and retained during and after the operation.

Acute inflammation of the gallbladder must always be differentiated from acute pancreatitis and perforation of an ulcer. The symptoms of acute inflammation of the gallbladder with empyema are not as severe as those of acute pancreatitis. The serum amylase test will very often help to clear this situation. At the present time, the surgeons should not operate on a patient with acute pancreatitis on account of the high mortality following this procedure. Acute inflammation of the gallbladder with empyema is in the same category. Watchful waiting, conservatively and scientifically performed, will save lives.

Another adjunct in the treatment before and after operation is the use of penicillin and streptomycin. As in other inflammatory conditions, penicillin will reduce the intensity of the acute inflammation. It will not reduce the size of the gallbladder which can only be accomplished by abstention from food and parenteral feeding. In every patient operated upon for gallbladder disease, and especially where a cholecystectomy has been performed, penicillin should also be administered postoperatively on account of the existence of the Welch bacillus in some patients.

The complications of perforation, gangrene and empyema of the gallbladder can all be avoided by an early prophylactic cholecystectomy before infection enters into the picture.

If an acute gallbladder is operated upon as an emergency case, the surgeon is apt to perform a cholecystostomy. This is a poor operation because the mortality following it is even a little greater than the operation of cholecystectomy. Furthermore, cholecystostomy as a preliminary to cholecystectomy requires two separate individual operations performed at an interval of two to four weeks. I have not performed a cholecystostomy in over fifteen

years. Cholecystectomy is the operation of choice and can always be performed after the acute symptoms have subsided. If the "open" operation of cholecystectomy is performed, there should be no danger from injury to the common duct or hepatic artery. In 99% of the cases, the operation is performed from below upwards. With this technic, the operation is more logical because the blood supply to the gallbladder is controlled. The structures can all be visualized if proper care is taken to expose the structures around the foramen of Winslow. Occasionally, one may be required to remove the gallbladder from above downwards. If the surgeon feels safer performing a cholecystectomy in this manner, this is the way he should do it.

CONCLUSIONS

1. Acute empyema of the gallbladder should not be operated upon as an emergency case.

2. The ordinary laboratory tests should be used before any operation is contemplated.

3. *The patient must be hospitalized, preferably on the surgical service.*

4. *All fluids by mouth must be avoided and parenteral feeding substituted.*

5. Most perforations and gangrene of the gallbladder result from inefficient treatment.

6. Over 95% of acute gallbladders subside under the proper treatment.

7. Liver deaths can also be avoided under this regime.

8. In patients with varicose veins past the fourth or fifth decade, ligations of these structures should be performed. These ligations and the abdominal work can be done simultaneously provided the surgeon has a proficient team in the operating room.

9. Palpation alone can determine the stage of the disease, and watchful waiting scientifically performed results in no danger.

10. Penicillin and at times streptomycin, should be used before and after operation.

11. It must be impressed upon the internist that acute empyema is not a medical disease.

12. There is no question that cholecystectomy is preferable to cholecystostomy. Where one operation can be performed successfully, we should not subject the patient to two operations.

255 South 17th Street.

Women's Club Opposes Health Plan.

Delegates to the annual convention of the General Federation of Women's Clubs, which has a membership of five million in the United States, recently adopted a resolution which opposed "government control of health services" and supported "the extension and development of voluntary health insurance."

The resolution, which strongly opposed Mr. Truman's health plan, carried great import because it expressed the feelings of millions of women on the matter of sickness taxation. The General Federation is made up of 16,500 clubs with a total membership 7,700,000. Only 2,700,000 of those members reside in foreign countries.

When the final action was taken there were only a few dissenting votes. It appeared as though every delegate stood up to be counted when the question was asked: "All those in favor of the resolution, please stand?"

Treatment for Whooping Cough.

Scientists of the Public Health Service, Federal Security Agency, believe they may be on the way to developing a more effective treatment for whooping cough, according to a report contained in the May 13 issue of *Public Health Reports*, a Service publication.

Current studies indicate that aureomycin, a relatively nontoxic antibiotic which can be given by mouth, may shorten the course of the disease and lessen its intensity. Drs. Joseph A. Bell, Margaret Pittman, and Byron J. Olson are the investigators working on this project in the Microbiological Institute, one of the National Institutes of Health at Bethesda, Maryland.

The early favorable results of the laboratory experiments justified a clinical investigation which was promptly undertaken. So far, 20 patients have been treated in their homes with aureomycin, with apparently beneficial results.

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THE CANCER COMMITTEE MEDICAL SOCIETY OF VIRGINIA

Case Reports

A 52 year old woman who had experienced no vaginal bleeding since a subtotal hysterectomy performed four years previously, began to spot. Fearful of cancer, she at once consulted her physician. He sought to relieve her of her cancerphobia and for the next two years, without even performing a pelvic examination, treated her with estrogens. The spotting gradually became more profuse and she finally visited another doctor. Pelvic examination revealed an ulceration of the left cervical lip. The biopsy report was squamous cell carcinoma, grade 2. Proper treatment was at once instituted at a tumor clinic.

A 53 year old woman who had passed the menopause five years before, developed vaginal bleeding. She consulted her doctor who, without doing a pelvic examination, prescribed some "white pills" (ergotrate?). Intermittent spotting persisted for four months, then there was a profuse hemorrhage. The same physician, still without doing a pelvic examination, prescribed more pills. When the severe bleeding persisted, the patient asked the advice of another physician. He performed a pelvic examination and found a League of Nations IV carcinoma of the cervix. Biopsy was reported squamous cell carcinoma, grade 3. He referred her to a tumor clinic.

A 47 year old woman who was still menstruating regularly, noticed some intermenstrual spotting. At the next period, she had a profuse hemorrhage. Her physician, without doing a pelvic examination, gave her "some medicine to check the bleeding." She got along all right for the next two months, then had a prostrating hemorrhage. Her physician referred her to a gynecologist who did a pelvic examination and found an eroding carcinoma of the cervix, League of Nations II, squamous cell, grade III.

COMMENT: 1. In all of the above cases, the patients' disease remained unrecognized and untreated for a significant period after the patients had reported to their physicians symptomatology which called for immediate, complete investigation.

2. These cases, and the many similar ones coming through our Tumor Clinics, teach a fundamental lesson which cannot be stressed too strongly—*abnormal vaginal bleeding must not be treated on a presumptive diagnosis. The cause must be found and the diagnosis established before any thought is given to treatment.*

3. In all three of these cases, the diagnosis could have been established immediately by what the physician should do every time he is presented with the problem of abnormal vaginal bleeding—a careful pelvic examination with visual inspection of the cervix and biopsy of any suspicious areas. Do not omit a pelvic examination because a patient is bleeding. The bleeding increases the chance of finding the source.

4. The Bureau of Cancer Control, State Health Department, Richmond, offers a tissue diagnostic service to every physician in Virginia. Even the containers for mailing specimens are supplied. Every physician can do biopsies as freely and obtain diagnoses almost as promptly as if he were practicing in a large hospital.

Color Televised Surgical Operations.

For the first time in history, surgical operations were seen on May 31st by means of natural color television. The color transmissions took place at the Hospital of the University of Pennsylvania bebefore members of the press, medical students and the hospital staff. This new development will enable large groups of medical students to study, close up in full color detail, surgical techniques and medical procedures which now can be so viewed by only a few at a time.

The color television equipment was specially designed and manufactured by Columbia Broadcasting System, Inc., for teaching surgery and medicine. Zenith Radio Corporation and Webster-Chicago Corporation collaborated with CBS in respect to the design and manufacture of the receivers. The first equipment of its kind to date—it is owned by Smith, Kline & French Laboratories—and is being demonstrated by them and later on at other medical conventions throughout the country as a contribution to medical teaching.

MEDICAL SOCIETY OF VIRGINIA

PUBLIC RELATIONS

HENRY S. JOHNSON, *Director*

Medical Society Entertains Congressmen.

On Monday evening, May 16, twelve officials of the Medical Society of Virginia had as dinner guests of the Society, Members of the House of Representatives and Senate of Virginia, at the Carlton Hotel, Washington, D. C.

The purpose of this meeting was obviously to become better acquainted with Virginia's representation in Washington and to better acquaint them with problems confronting our people and the medical profession.

It is equally obvious that the advice of Virginia's representation as to how best to proceed in the future, should be solicited.

Dr. M. Pierce Rucker, President of the Society opened the meeting with a statement on current interest referring to problems and possible solutions. He indicated that government or political medicine was of ultimate concern to the public and that the best interest of all could be served only by a full understanding of the subject with respect to the past, present and future.

Each doctor present was introduced and given an opportunity to express his views.

Dr. Joseph S. Lawrence, in charge of the Washington Office of the American Medical Association made an interesting talk.

Dr. James McVey, Chairman of the Council on Medical Service of American Medical Association, St. Louis, Missouri, made a splendid contribution to the conference.

Dr. H. B. Mulholland, Charlottesville, referred to the aims of the medical profession and progress that was being made in realizing these objectives.

Dr. William R. Pretlow of Warrenton, discussed voluntary pre-payment medical care and hospitalization, their contribution and some of their problems.

Dr. John T. T. Hundley, Lynchburg, told of the purposes of the national educational campaign and referred to its progress in Virginia.

Dr. John Hazel, Arlington, told of the interest in his medical society and related a number of interesting activities.

Senator A. Willis Robertson of Virginia, presided over the rest of the meeting, introducing each member of Virginia's representation present, and the group had at least a ten to fifteen minute talk from each one.

Dr. Morrison Hutcheson of Richmond, summed up the discussion of the meeting and referred to the values of such a get-together.

Since that time, two excellent references to the meeting have been written by Dr. Russell Buxton, Newport News, and Dr. John T. T. Hundley, of Lynchburg.

Letters of appreciation from every representative present have come to the Medical Society of Virginia and although it was a social function, it was a most constructive affair.

Colorado Doctors Solve Own Problems.

A few years ago, it became evident to the Colorado State Medical Association that many patients and members of the lay-public felt that they had grievances against the practice of medicine in that state.

This appeared to be of sufficient significance to the leadership in the society to justify the setting up of the necessary machine to deal with this.

The official committee named to handle this work was "The Board of Supervisors". Although it should be borne in mind that the responsibilities and functions of this committee included matters, whom some might regard as problems of ethics, apparently the most important work of this committee was regarded to be in the interest of "cleaning their own house" of criticisms, whether justified or not, aimed at the medical profession.

After several years of operation, officials of the Colorado State Medical Association point with pride to the excellent results obtained by this system.

They point out that whereas only a small percentage of complaints being handled by this committee are found to be justifiable grievances, the existence and work of the committee constitutes a valuable

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instrument in establishing confidence on the part of the public in the medical profession and also proved valuable to the profession itself.

Their committee meets regularly once a month, dealing with principles, leaving references to the individual as a last resort; but it is indicated that severe action has been taken in several instances and which has inured to the best interests of medical care and the medical profession in that state.

J. M. EMMETT, *Chairman*
Public Relations Committee

General Practitioner of the Year.

There has been so much recent interest throughout the country in the General Practitioner chosen for the year, that it is believed to be important that this selection not be overlooked in 1949.

Perhaps the procedure of nominations in the local medical societies or through the selection of the practitioner of the state, will be out-lined by the committee in the American Medical Association headquarters on this, or instructions will be sent from your state society in the near future.

In view of the fact that a number of local medical societies in Virginia still hold rather infrequent meetings, it is suggested that no one local society in Virginia overlook the opportunity of nominating a candidate for this honor.

MISCELLANEOUS

American Medical Association Assessment For Educational Campaign.

Lists of contributors to the special assessment for the educational campaign of the American Medical Association as published in the March, April and May issues of the *Monthly* gave the total contributors from Virginia as 1185. The following who have contributed through June 10th makes the total from Virginia as 1357:

Albemarle
Dr. A. M. Smith
Dr. H. L. Smith, Jr.

Alexandria
Dr. S. H. Williams
Dr. J. D. Kernodle

Alleghany-Bath
Dr. Michael I. Hanna
Dr. J. O. Easley

Arlington
Dr. Lloyd B. Burk
Dr. Joseph D. Casolaro
Dr. Jerome A. Cope
Dr. Hermann F. Diamant
Dr. William D. Dolan
Dr. John H. Gilligan
Dr. M. W. Glover
Dr. John T. Hazel
Dr. Louisa C. Littleton
Dr. Robert B. Mallett
Dr. Eugenia E. Murphy
Dr. A. R. MacPherson
Dr. M. Roy Nicholson
Dr. Robert L. Norment
Dr. K. N. Ostergard
Dr. Robert E. Pagan
Dr. Alfred M. Palmer
Dr. Michael A. Puzak
Dr. June C. Shafer
Dr. Leo Solet
Dr. Blanche Tabor

Buchanan-Dickenson
Dr. Theodore C. Andrew

Danville
Dr. W. C. Yeatts

Fairfax
Dr. Lawrence A. Jacklin
Dr. T. B. McCord
Dr. Emanuel Newman
Dr. B. F. Phillips
Dr. Nelson Podolnick
Dr. J. D. Zylman

Fauquier
Dr. James L. Dellinger

Fredericksburg
Dr. John E. Cole

Fourth District
Dr. Leta J. White
Dr. G. N. Carter

Lynchburg
Dr. Elizabeth Edmunds

Mid-Tidewater
Dr. A. W. Lewis, Jr.
Dr. T. E. Smith
Dr. F. B. Wilson

Norfolk
Dr. M. S. Andrews
Dr. G. R. Berkeley
Dr. M. H. Bland
Dr. W. M. Bland
Dr. J. H. Bocock
Dr. C. P. Brown
Dr. Mary T. Bunting
Dr. A. A. Burke
Dr. Julius Caplan
Dr. James E. Diehl
Dr. J. C. Dunford
Dr. H. W. Fink
Dr. C. D. Freeman
Dr. A. W. Glaess
Dr. W. S. Hadley
Dr. G. P. Hand, Jr.
Dr. Harry Harrison
Dr. M. S. Herrington
Dr. John H. Hill
Dr. A. G. Horton
Dr. K. W. Howard
Dr. T. Elmore Jones
Dr. Otto Kastenbaum
Dr. Anne L. Lee
Dr. M. H. Legum
Dr. J. R. Leigh
Dr. Southgate Leigh, Jr.
Dr. Edward D. Levy
Dr. Bernard Lidman
Dr. C. H. Lupton
Dr. Lemuel Mayo
Dr. L. A. McAlpine
Dr. W. P. McDowell
Dr. M. J. Miller
Dr. F. N. Mullen, Jr.
Dr. R. B. Nicholls
Dr. Arthur D. Parker
Dr. Nelson S. Payne
Dr. S. B. Pope
Dr. John A. Proffitt
Dr. C. N. Psimas
Dr. B. H. Raymond
Dr. John O. Rydeen
Dr. J. T. St. George
Dr. Milton Salasky
Dr. E. Schweiger
Dr. Ben Steingold
Dr. Arnold F. Strauss
Dr. Harry Sutelan
Dr. John A. Vann
Dr. Cary E. Via
Dr. H. A. Wall
Dr. C. F. Ward
Dr. D. P. West
Dr. S. B. Whitlock
Dr. J. B. Woodley

Northern Neck
Dr. R. E. Booker
Dr. P. P. Deming

Northern Virginia
Dr. G. G. Crawford

Patrick-Henry
Dr. Homer H. Price

Richmond
Dr. R. L. Bailey
Dr. J. B. Black, Jr.
Dr. M. L. Boyle
Dr. D. Coleman Booker
Dr. Harry Brick
Dr. Manfred Call
Dr. R. W. Crossen
Dr. W. A. Eskridge
Dr. H. T. Garriss
Dr. Fleming W. Gill
Dr. Thomas F. Gill
Dr. W. C. Grinnan
Dr. William R. Hill
Dr. W. H. Higgins
Dr. Oscar L. Hite
Dr. Thomas E. Hughes
Dr. Francis B. Lee
Dr. James C. LeFon
Dr. Carolyn M. McCue
Dr. Howard McCue
Dr. H. H. McGuire
Dr. R. C. Manson
Dr. J. D. Markham
Dr. Blake W. Meador
Dr. Barney Plotnick
Dr. W. D. Poe
Dr. Edward S. Ray
Dr. H. M. Richardson
Dr. W. W. Rixey
Dr. H. W. Rollings
Dr. E. B. Shepherd
Dr. L. W. Snead
Dr. E. B. Talbot
Dr. D. D. Talley
Dr. Elam C. Toone
Dr. E. T. Trice
Dr. H. St. G. Tucker
Dr. J. T. Tucker
Dr. A. E. Turman
Dr. J. L. Ulmer
Dr. Edwin Vaughan
Dr. G. A. Welchons
Dr. L. J. Whitehead
Dr. E. S. Williams
Dr. H. M. Wingfield
Dr. I. S. Zfass

Roanoke
Dr. J. H. Bailey
Dr. Lula W. Garst
Dr. G. G. Gooch
Dr. Faith F. Gordon
Dr. W. Banks Huff
Dr. A. M. Jacobson
Dr. B. C. Moomaw
Dr. R. S. Owens, Jr.
Dr. R. A. Richards
Dr. A. D. Shapiro

Southampton
Dr. H. L. Gardner
Dr. C. F. Gryte
Dr. K. Hirsch
Dr. W. D. Varner

Southwest Virginia
Dr. Don L. Greever
Dr. William N. Greever
Dr. R. H. Harrington
Dr. M. C. Newton
Dr. S. H. Yokeley

Tazewell
Dr. G. D. Vermilya

Warwick
Dr. J. E. Battalia
Dr. Thomas N. Hunnicutt

Wise
Dr. Edward B. Blum

BOOK ANNOUNCEMENTS

Handbook of Materia Medica, Toxicology, and Pharmacology. For Students and Practitioners of Medicine. By FORREST RAMON DAVISON, B.A., M.Sc., Ph.D., M.B., Consultant and Toxicologist, Minneapolis, Minnesota; formerly assistant professor of pharmacology in the School of Medicine, University of Arkansas; Medical Department, The Upjohn Company, etc. Fourth Edition. St. Louis, The C. V. Mosby Company, 1949. 730 pages. With 35 illustrations, including 4 in color. Cloth. Price $8.50.

The Compleat Pediatrician. Practical, Diagnostic, Therapeutic and Preventive Pediatrics. By W. C. DAVISON, Professor of Pediatrics, Duke University School of Medicine. Sixth Edition. Duke University Press, Durham, North Carolina, 1949. Cloth. Price $4.75.

Medicine Throughout Antiquity. By BENJAMIN LEE GORDON, M.D., Member, American Association of the History of Medicine and American Academy of Ophthalmology and Otolaryngology; attending Ophthalmologist to Shore Memorial Hospital, Somers Point, New Jersey, and to Atlantic County Hospital for Mental Diseases, Northfield, New Jersey; etc. Foreword by Dr. Max Neuburger. Philadelphia, F. A. Davis Company, 1949. xvii-818 pages. Cloth. Price $6.00.

How to Become a Doctor. A Complete Guide to the Study of Medicine, Dentistry, Pharmacy, Veterinarian Medicine, Occupational Therapy, Chiropody and Foot Surgery, Optometry, Hospital Administration, Medical Illustration, and the Sciences. By GEORGE R. MOON, A.B., M.A., Examiner and Recorder, University of Illinois, Colleges of Medicine, Dentistry and Pharmacy. The Blakiston Company, Philadelphia, 1949. ix-131 pages. Cloth. Price $2.00.

Outwitting Your Years. By CLARENCE WILLIAM LIEB, M.A., M.D. Prentice-Hall, Incorporated, New York, 1949. viii-278 pages. Cloth. Price $2.75.

Help Yourself to Better Sight. By MARGARET DARST CORBETT. Prentice-Hall, Incorporated, New York, 1949. xiv-218 pages. Cloth. Price $2.50.

Essentials of Pathology. By LAWRENCE W. SMITH, M.D., F.C.A.P., Formerly professor of pathology, Temple University School of Medicine; Formerly assistant professor of pathology, Harvard Medical College, etc. and EDWIN S. GAULT, M.D., F.C.A.P., Associate professor of pathology and bacteriology, Temple University School of Medicine. Third Edition. The Blakiston Company, Philadelphia. 1948. 764 pages. 740 illustrations. Cloth. Price $12.00.

The essentials of pathology are herein presented by the study of case reports interpreted in the light of gross pathologic data and roentgenograms when pertinent and analyzed by the methods of histopathology. The method is an admirable one, but the bibliography and the various subdivisions of chapters and sections must necessarily keep up, and in step with, the current literature.

Thus under the "Lymphoid and Reticulo-endothelial Tumors", it would be well to cite Gall and Mallory's paper on the "Malignant Lymphoma" of 1942, even at the expense of omitting Gall's lesser paper of 1941. The "Malignant Lymphoma" are discussed in three pages with one case report which is followed by a discussion of "Lympho-epithelioma" and then follow five pages on "Hodgkin's Disease" and "Hodgkin's Sarcoma", and "Reticulo-endothelial Sarcoma". Only "Giant Follicle Lymphoma", "Lymphocytoma", and "Lymphoblastoma"

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thus appear under the main heading "Malignant Lymphoma". This type of arrangement does not help to clarify the neophytes conception of "lymphoma" and it might be well to mention the more recent classifications and their advantages and limitations.

The selection of clinical cases to illustrate certain conditions could in some instances be improved upon. On page 298 to depict a case of "medulloblastoma", the authors chose a 34 year old woman whose symptoms began 7 years before and in which a diagnosis of pituitary tumor was made and 32 x-ray treatments were given without benefit. The age of the patient, the neurological findings given, the history, and the lack of response to irradiation were in no way typical of the average case of medulloblastoma from a purely "case teaching" point of view. On the other hand, the discussion and case report on "chordoma" seem to be extremely well chosen.

The book is, like most works on pathology, not without its "index" difficulties. Thus the subject of "pheochromocytoma-chromaffinoma" is considered on pages 299, 306, 450 and 638 with only two pages noted under each title in the index with a great deal of interesting information still not included on any of the pages listed.

Errors in the labelling of roentgenograms and identification of case reports do occur. Thus on page 251, Fig. 255, X-ray-*arm*: actually is a *forearm* and Case 63 fails to mention any involvement of the forearm, in the clinical history. The multiple bone lesions mentioned in the history, and the roentgenogram depicted are not at all convincing for the heading "Bone: Benign Giant Cell Tumor" nor can we agree with the text that it "tends to occur at the diaphyseal ends of the long bones." Jaffé's work is mentioned but his three types of giant cell tumors are not clearly outlined in the text.

In general, the authors have carried out their "case history" method with unusual success. To incorporate the rapid and tremendously changing literature of "Pathology" into a case book for sophomore medical and dental students is indeed, a monumental and never ending task.

F. B. MANDEVILLE

New Books.

The following books are recent additions to the Library of the Medical College of Virginia, and are available to our readers under usual library regulations:

Addis—Glomerular nephritis.
Alford—Cerebral localization: outlines of a revision.
Bard—The doctor wears three faces.
Basic science for orthopedics.
Bradley—Pathological physiology of uremia in chronic Bright's disease.
Brodal—Neurological anatomy in relation to clinical medicine.
Gogan—Neurology of the ocular muscles.
Conn—Current therapy.
Chopra—Indigenous drugs of India; their medical and economic aspects.
Dressler and Roesler—An atlas of electrocardiography.
Encyclopaedia Brittanica. New 1949 ed.
Eppinger—Die permeabilitaes pathologie.
Fuch—Atlas of ocular fundi.
Glick—Techniques of histo- and cytochemistry.
Grassé—Traité de zoologie. Vol. XI.
Helmholtz—On thought in medicine.
Henle—On miasmata and contagia.
Kabat and Mayer—Experimental immunochemistry.
Krantz and Carr—Pharmacologic principles of medical practice.
Levine and Harvey—Clinical auscultation of the heart.
Lockhart—Living anatomy.
Modern drug encyclopedia and therapeutic index.
Nungester—An illustrated study aid for medical bacteriology.
Rowe—Elimination diets and patients allergies. 2d ed.
Singer—Short history of medicine.
Virginia State Health Department—Survey of hospital facilities in Virginia.
Von Weizsacker—Der Gestalt Kreis.
Witmer—Psychiatric interviews with children.

PUBLIC HEALTH

L. J. ROPER, M.D.,
State Health Commissioner of Virginia

The report of the Bureau of Communicable Disease Control of the State Department of Health for May 1949 as compared with the same month in 1948, and for the period of January through May 1949 compared with the same period in 1948, is as follows:

	May 1949	May 1948	Jan.-May 1949	Jan.-May 1948
Typhoid and paratyphoid	8	12	31	53
Diarrhea and dysentery	186	205	1,285	1,393
Measles	3,416	1,478	18,837	4,192
Scarlet fever	41	76	358	507
Diphtheria	9	16	61	99
Poliomyelitis	1	3	9	9
Meningitis	6	9	46	46
Undulant fever	6	10	25	39
Rocky Mountain spotted fever	10	5	11	5
Tularemia	1	4	8	24

ROCKY MOUNTAIN SPOTTED FEVER

With the recurrence of the tick season and the possibility of infection with Rocky Mountain spotted fever it may be useful to review our knowledge of its etiology, incidence, treatment and control measures employed.

Incidence:

Table I shows the number of cases of Rocky Mountain spotted fever reported in Virginia since 1931 and the deaths for this same period.

TABLE I

	1931	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48
Reported Cases	6	9	23	47	45	50	54	72	50	46	33	47	56	81	99	93	67	64
Deaths	0	1	4	8	10	9	9	13	5	7	11	8	16	14	21	14	10	9

Up to May 28, 1949, 11 cases had been reported in Virginia this year, 10 in May and 1 in April. There have been no deaths.

Table II shows the monthly distribution of the 64 cases of Rocky Mountain spotted fever reported in Virginia in 1948.

TABLE II

January	February	March	April	May	June	July	August	September	October	November	December
				5	17	17	19	2	4		

The monthly distribution of reported cases follows the pattern of previous years with the highest incidence in June-July-August.

Since 1941, Rocky Mountain spotted fever has been reported from 88 counties in the State. During this period no reports have come from the other 12 counties: Bath, Bland, Craig, Floyd, Giles, Highland, Mathews, Northumberland, Pulaski, Surry, Washington, and Westmoreland.

The fatality rate is somewhat lower in the Eastern than in the Western States. In the 10 year period (1930-39) the crude fatality rate for Idaho and Montana was 28.1% while for Maryland and Virginia it was 18.4%.

Etiology:

In Virginia the American dog tick (Dermacentor variabilis), also locally called the wood tick, is probably the only vector of spotted fever. The adult males and females are oval in shape, almost ½ inch in length and brown in color. The females are darker and have a larger area of lines, hairs and other markings just behind the "head".

Diagnosis:

From the standpoint of laboratory assistance, a diagnosis of active Rickettsial infection cannot be made by serological test alone. Diagnosis can be arrived at only by correlation of laboratory findings and clinical picture. There is no single positive titer, either in the complement fixation test or the Weil-Felix, which is diagnostic of present infection. A *rising* titer is very strong presumptive evidence of present active Rickettsial infection. For this reason it is very important to obtain two or

more successive specimens of serum from every patient, seven to ten days apart. The first specimen should be taken from the fifth to the tenth day of illness.

Control and Treatment:

Protection from Rocky Mountain spotted fever through vaccination lasts approximately one year and unless one is going to spend time in known infected areas, vaccination is not recommended. Vaccination is recommended for those whose work or play takes them into the woods for prolonged periods—highway workers, loggers, campers. Personal protective measures such as proper clothing and daily inspection for ticks are advisable.

In the treatment of Rocky Mountain spotted fever Aureomycin has proved a spectacular remedy—almost a specific. Cases with high temperature and characteristic rash have been rendered afebrile after 48-72 hours of intensive treatment with this new antibiotic.

United States Has Lowest Rates.

Final tabulation of births and maternal deaths for 1947 by the National Office of Vital Statistics indicates a new record low maternal mortality rate of 1.3 per thousand live births, according to an editorial in a recent issue of the *Journal of the American Medical Association.*

No other Nation has reported a lower rate.

In 1933 the American rate of 6.2 placed this country eleventh among the leading Nations. Since then the drop to 1.3, amounting to a 79% reduction, has "undoubtedly raised the rank of the United States to first place or close to first place" according to the Journal.

"The phenomenal reduction in maternal mortality in the United States," said the Journal, "has not been restricted to any single State or group of States, but has been relatively uniform throughout the entire country."

None of the States in 1947 had a rate above 2.6. Connecticut, Delaware, Iowa, Massachusetts, Minnesota, Oregon, Rhode Island, Utah, and Wyoming reported a rate less than 1.0, with Minnesota the lowest at 0.6.

None of the States had a rate lower than 4.3 in 1933. In that year the spread between the highest and lowest was from 11.5 to 4.3, or 7.2. In 1947 the spread dropped to 2.0.

The great improvement in the rate was reflected among non-whites as well as white Americans. For whites the rate declined from 5.6 to 1.1 and for non-whites from 9.7 to 3.3.

"It is also abundantly clear," stated the *Journal,* "that this form of health progress has been extremely general throughout the United States during these 15 years."

WOMAN'S AUXILIARY TO THE MEDICAL SOCIETY OF VIRGINIA

President—MRS. R. M. REYNOLDS, Norfolk
President-Elect—MRS. C. E. HOLDERBY, Hilton Village
Recording Secretary—MRS. T. S. CHALKLEY, Richmond
Corresponding Secretary—MRS. CHARLES LUPTON, Norfolk
Treasurer—MRS. REUBEN SIMMS, Richmond
Parliamentarian—MRS. H. W. ROGERS, Norfolk
Historian—MRS. M. H. HARRIS, West Point

Warwick County Auxiliary.

At the April luncheon meeting of this Auxiliary, Miss Virginia Fox, educational director of the Elizabeth Buxton Hospital School of Nursing, presented a film on cancer "The Doctor Speaks His Mind".

At the meeting on May 25th, Mrs. Chester D. Bradley, President, selected the following committees for the annual meeting of the State Auxiliary to be held at Old Point Comfort, October 9-12: Reception: Mrs. William A. Read and Mrs. Frederick Nimrod Thompson; Registration: Mrs. Guy C. Amory and Mrs. J. W. Carney; Flowers: Mrs. Russell Von Lehn Buxton; Transportation: Mrs. William A. Mitchell and Mrs. Paul Hogg; Entertainment: Mrs. Barnes Gillespie and Mrs. Louis Richman. Mrs. Thomas N. Hunnicutt, Jr., will give the address of welcome.

EDITORIALS

Sir William Osler (1849-1919)*

ONE hundred years ago Bond Head was on the edge of the Canadian Wilderness. The Rev. Featherstone Osler's home was the social center. Chippewa Indians often paid migratory visits and the "stump farmers" of the neighborhood gathered on all occasions. Every 12th of July the Orangemen with their sashes and rosettes, gathered at the Corners and marched the mile to the parsonage, where speeches were made and felicitations offered in return. On July 12, 1849, the 8th Osler child was born. His name was to have been Walter Farquhar, but later in the day when he was shown to the Orangemen, they dubbed him young Prince of Orange and insisted that he be named William. Thus a future Regius Professor of Medicine of Oxford University was ushered into the world on the American frontier.

What wonderful changes in medicine this young Canadian saw and brought about. After a good classical education, Osler studied medicine at Toronto and at McGill. At neither place were there laboratories, but at McGill the students had the run of the Montreal General Hospital. Upon graduation in 1872 Osler went to London and worked under Burdon Sanderson in Physiology. Later he went to Berlin and worked under Virchow and to Vienna where he took a number of courses. He returned to Canada in the summer of 1874. The following September he was appointed lecturer of the Institutes of Medicine, which included Physiology and Pathology and everything that was taught with a microscope. The next year he ordered 12 microscopes, the first at McGill, which he paid for out of his own meagre salary. His first clinical work was in the smallpox wards of the Montreal General Hospital. It was not long before he was doing all the autopsies. Four years later he succeeded Dr. Drake as physician to the Montreal General Hospital.

In 1884 Osler was called to the University of Pennsylvania. Pennsylvania, following the lead of Harvard, had just adopted a three year curriculum. Osler introduced bedside teaching. Five years later he went to Baltimore as Physician-in-Chief of the Johns Hopkins Hospital. Here in Baltimore, he, together with Dr. William H. Welch and others of that wonderful first Faculty, developed a new medical school untrammelled by traditions. The result was a Medical University with the best features of German and English medical teaching—laboratories, bedside teaching, and long term residencies.

Finally, in 1905 he received a call to Oxford to succeed his old teacher, Burdon Sanderson. He, himself, looked on the move as a kind of retirement.

Wherever he went—Montreal, Philadelphia, Baltimore or Oxford—he was a medical catalyst. He improved medical instruction, stimulated medical societies for graduates, as well as undergraduates. Medical publications took on new life. He activated libraries, and stimulated public interest in health matters. No one has done more for American Medicine.

*This is the third of a series "Doctors for whom the public should be thankful."

The Influence of Two Virginia Cesarean Sections

WHEN the knowledge obtained through a new discovery is added to the great pool of common knowledge it may stimulate further advances in the same line or even in entirely unrelated fields. Such knowledge may spread in one of three ways (1) personal contact in ordinary every day relations (2) publication (3) pressure actuated by desire for either gain or fame. In the first instance the spread is slow

and limited in extent. When a report is made in a magazine the knowledge is disseminated more rapidly, depending upon the character of the journal and its circulation. Oliver Wendell Holmes' original communication on the contagiousness of puerperal fever attracted no immediate attention because it was published in a short-lived New England magazine that had only local subscribers. The pressure method, so common in modern advertising, is seldom used in medicine, although the controversy incident to the discovery of anesthesia is a notable exception.

Two Cesarean operations performed in Virginia about the beginning of the Nineteenth Century afford good examples of the first two methods of the dissemination of new knowledge. In 1794 Dr. Jesse Bennett of Rockingham County, Virginia, performed a Cesarean Section upon his own wife and removed both ovaries. Dr. Alexander Humphreys of Staunton was called in consultation and both he and Dr. Bennett failed to deliver the baby with forceps. Believing that she would die whether a craniotomy or a Cesarean Section were done, Mrs. Bennett chose the section. Dr. Humphreys refused to operate. Dr. Bennett operated and saved both mother and child. To make sure that he would not be subjected to such an ordeal again, he removed both ovaries. Dr. Joseph L. Miller was the first to describe this operation. (*Am. Med. His. N. S.* **10**:23,1938). Some three years after the operation Dr. Bennett moved to the Ohio River with his wife and daughter, where he developed a practice that extended over an area of 100 to 150 miles in diameter.

In 1827 Dr. John Lamberth Richmond performed what is usually considered the first Cesarean section in the United States. It was reported in the *West Journal of Med. & Phys. Sc.* **3**:485,1830. As it was performed at Newtown, Ohio, less than 85 miles from Mason County, Kentucky, where Dr. Bennett had been living for 30 years, it is likely that Dr. Richmond knew of Dr. Bennett's successful operation.

It is also likely that Bennett's operation had influence in a different direction. Dr. Ephraim McDowell studied medicine under Dr. Alexander Humphreys before he went to the University of Edinburgh. Dr. Hugh Trout is of the opinion that McDowell must have visited Humphreys when he returned to this country in the summer of 1794. If so, then they must surely have discussed the remarkable operation that Jesse Bennett had performed six months previously and a knowledge of this operation must have influenced him to his decision to operate when confronted with a case of ovarian cyst in 1809, a condition which up until then was considered hopeless. (*Am. Med. Hist. N. S.* **10**:168,1938.)

The second Virginia Cesarean Section was performed by an "irregular practitioner" of Fairfax County in 1828, It was reported by Dr. W. L. Weems in the *American Journal of Medical Science* (**8**:257,1936). Dr. Weems witnessed the operation but had no part in it. The fetus was in a half dissolved state. The mother died in the second week after the operation, her death being attributed to her eating animal food and drinking cider. The interesting feature in this case was that two or three sutures were placed in the uterus. Before this the uterus had been left unsutured. From this time on, more and more operators closed the uterine wound with sutures. In 1852, Frank E. Polin of Springfield, Kentucky, used silver wire for this purpose. A list of these operations was compiled by Robert P. Harris (*Am. J. Med. Sc.* **75**:313,1878). Dr. Nicholson J. Eastman (*Am. J. Obst. & Gynec.* **24**:919,1932) is of the opinion that these operations tremendously influenced Sänger whose classical report in 1882 marks the beginning of the modern period in the history of this operation.

Little did Dr. Jesse Bennett dream, when confronted by his fearful problem, that its successful solution would have any influence in the performance of one of the

world's great operations, one that became a mile stone in the history of surgery. While the "irregular practitioner" of Fairfax County remains unknown, his work is not forgotten.

Miss Agnes Edwards

ON May 9th, Miss Edwards had to stop work temporarily on account of a slight cerebral vascular accident. Fortunately the area involved was small and unimportant. She has now left the hospital and is recuperating rapidly. However, her physician wants her to rest for a couple of months. For the first time in many years the MONTHLY has been without her guidance.

The VIRGINIA MEDICAL MONTHLY was started in 1874 by Miss Edwards' father, the late Dr. Landon B. Edwards. Until his death in 1910, Dr. Edwards owned the magazine and ran it as a private venture. As his children grew up, the publishing of the MONTHLY (or SEMI-MONTHLY, for at one time it was published twice a month) became a family affair. We doubt if Miss Edwards, herself, knows when she first began working on the MONTHLY.

Upon Dr. Edwards' death, his son, Dr. Charles M. Edwards, became managing editor and continued as such until he entered the Service in 1918. In 1919, the Medical Society of Virginia purchased the MONTHLY and continued to publish it under the direction of a Publication Committee and Miss Edwards. Twenty-five years ago, Miss Edwards became, in addition, the executive secretary-treasurer of the Medical Society of Virginia. These two positions she has continued to fill to the satisfaction of every one.

In a real sense, Miss Edwards has grown up with the MONTHLY. It is a part of her life. She and the MONTHLY are inseparable. She has built for herself and the MONTHLY an enviable nation-wide reputation. The Council is thoroughly cognizant of how much the Medical Society of Virginia owes Miss Edwards for her long and faithful service. At its last meeting, it appointed a committee to arrange Miss Edwards' partial or full time retirement at her pleasure. The thought of the Council was that whenever Miss Edwards felt that the burden was too great, some satisfactory arrangement would be made. Miss Edwards' long experience is too valuable to lose entirely. Possibly it would have been better had the Council acted sooner. At any rate, we hope that Miss Edwards will soon be back at work in her full capacity or at least for a short time each day.

Floral Eponym

Zaluzianskyia

ZALUZIANSKY, ADAM, FL.1592

ZALUZIANSKY was a Polish physician and botanist. He was born in the middle of the XVI Century and became a distinguished professor of medicine in the University of Prague. By some, he is said to have discovered the sex of plants, but that is an error, for in his time knowledge of this subject was scarcely more advanced than that of the ancients. His principal works are: *Methodi rei herbariae libri tres,* Prague, 1592; *Harmonia confessionum orthodoxarum regni* Bohemiae, Prague, 1609; Animadversiones in Galenum et Avicennam.

Zaluzianskyia, night phlox, is a genus of twenty species of fragrant South African plants, most of which are night blooming.

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SOCIETIES

The Southwest Virginia Medical Society

Held its Spring meeting on May 26th at the Hotel Roanoke, Roanoke, under the presidency of Dr. R. M. DeHart, Radford. At the afternoon session, the following program was presented: Geriatric Dermatoses by Dr. William H. Kaufman, Roanoke; and Acute Poisoning in Children by Dr. Jay M. Arena, Duke University, Durham, North Carolina. Dr. John T. T. Hundley, Lynchburg, State Education Chairman, and Mr. Henry S. Johnson, Public Relations Director, Medical Society of Virginia, gave some highlights on Compulsory Health Insurance. The Society went on record as being opposed to Compulsory Health Insurance. Following a business session, social hour, and banquet, Dr. Robert N. Cradick, Duke University, Durham, North Carolina, spoke on Psychosomatic Gynecology, and Dr. William N. Nicholson, also of Duke University, on Some Neurologic and Vascular Complications of Diabetes Mellitus.

The Fall meeting of this Society will be held in Radford the first Thursday in October, at which time one of the guest speakers will be Dr. Karl J. Karnaky of Baylor University, Houston, Texas, who will speak on Endometriosis.

Dr. Andrew F. Giesen, Radford, is secretary-treasurer of this Society.

The Fauquier County Medical Society

Held its regular monthly meeting on May 19th, with Dr. V. L. McCullers presiding.

The program consisted of interesting discussions on community health problems and on the status of workers and their freedom in choice of physicians under existing compensations laws.

Dr. William R. Pretlow was elected delegate and Dr. V. L. McCullers alternate delegate to the Medical Society of Virginia meeting to be held in October, at Old Point Comfort, Virginia.

Election of officers was accomplished with Dr. M. B. Hiden being chosen as President for the coming year. Other officers elected were: 1st Vice President, Dr. Charles Martin of Manassas; 2nd Vice President, Dr. O. L. Huffman of Marshall; Secretary and Treasurer, Dr. James L. Dellinger of Warrenton. A vote of thanks was given the retiring officers for their work during the past several years.

The meeting was concluded with a short scientific program consisting of the showing and discussion of x-rays on interesting orthopedic cases.

JAMES L. DELLINGER

Norfolk County Medical Society.

At the annual meeting of this Society, the following officers were elected: President, Dr. Frank P. Smart; president-elect, Dr. George Schenck; vice-president, Dr. Brock D. Jones; secretary, Dr. Walter P. Adams; and treasurer, Dr. J. Franklin Waddill. The last two were re-elected.

Augusta County Medical Association.

The regular meeting of this Association was held on May 4th at the General Wayne Hotel in Waynesboro. Dr. Elmer L. Sevringhaus gave a very interesting talk on "Therapy with Estrogens and Progesterone in Disorders of the Female Reproductive System". He is Director of Clinical Research of Hoffman-LaRoche, Nutley, New Jersey, and is considered an authority on this subject. Dr. Charles Putney of Staunton read a paper on "Socialized Medicine". Delegates and alternates to the annual meeting of the State Society were elected at this time.

CHARLES F. GAYLORD, *Secretary*

The Tazewell County Medical Society

Held its regular bi-monthly meeting on April 21st in Tazewell, with Dr. G. D. Vermilya, president, presiding. Dr. L. L. Thompson, Richlands, read a paper on Alimentary Dysglycemia. Dr. F. E. LaPrade, Jewell Ridge, was elected to membership, and Dr. Mary Elizabeth Johnston, Tazewell, was named delegate to the State meeting in October.

Northampton County Medical Society.

Dr. J. M. Lynch, president of this Society, has appointed Dr. John R. Mapp, Nassawadox, as secretary-treasurer, to succeed Dr. Charles Garcia who is now in the Medical Corps of the U.S.A.F. at Langley Field. The next meeting of this Society will be a picnic on July 14th.

NEWS

Annual Meeting of the Medical Society of Virginia.

The ghosts of the past are walking at Old Point Comfort! After 69 years the Medical Society of Virginia is coming back to the famed tourist resort on the shores of Hampton Roads and Chesapeake Bay. Many changes have come over our nation and our profession since the meeting of 1881. The doctors who met in the Hygeia Hotel (predecessor to the Chamberlin) at Old Point Comfort were only 16 years removed from the bitterest and bloodiest war of all history up to that time. Many had served in it with distinction. The president's chair was occupied by Dr. Hunter Holmes McGuire of Richmond in whose arms Stonewall Jackson died after the battle of Chancellorsville. Only a short walk from the hotel was grim old Fortress Monroe in which Jefferson Davis had been imprisoned for several years after the war. The Confederate chief was still alive and had just published that same year of 1881 his two-volume history, "The Rise and Fall of the Confederate Government."

In 1881 the medical profession was just entering upon that great era of discoveries and advances in both medicine and surgery which has continued down to the present day. The typhoid bacillus had been discovered in 1880 and the tubercle bacillus was to be discovered in 1882. Max Saenger of Leipzig was to make known his improved cesarean section in 1882, but James Thacker Boutelle of Hampton, Virginia, had already closed the uterine incision with three silk sutures in 1879. In spite of all this, therapy was still in its infancy. The mainstays were purging, poulticing, blistering, cupping and leeching.

In 1881 there was no Newport News and no Phoebus, only Hampton. Still the doctors attending the meeting at Old Point Comfort found plenty of recreation between the scientific sessions. The Commanding Officer of Fortress Monroe threw open the fortifications for their inspection. Carriages were procured to take the doctors over to Hampton Institute and the National Soldiers' Home just outside of Hampton where they were cordially entertained. Mr. Harrison Phoebus, the genial proprietor of the Hygeia Hotel, arranged a steamboat trip to Yorktown. A guiding light of the meeting was the venerable Dr. George William Semple of Hampton who was head of the Local Committee on Arrangements.

Such were the thoughts which went through the mind of your correspondent Wednesday evening, May 25 last, as he sat in a private dining room at the Chamberlin Hotel with the Local Committee on Arrangements headed by Dr. Paul J. Parker, Hampton, and Dr. Waverly R. Payne, Newport News. The entire medical profession of the Lower Peninsula was represented from Hilton Village to Newport News to Hampton to Phoebus. It is too early to tell you about the many treats we are preparing for you down in this showplace of the nation. Suffice it to say that if the shade of George William Semple was present that evening it must have been highly satisfied by the scope of our plans for your entertainment. George William Semple was not a man to do things by halves!

It is our goal to make the social side of the annual meeting a thing that you will remember for the rest of your life. Look what we have to work with! Your meeting is set in a showplace which is a play place—the historic yet sumptuous Chamberlin Hotel, the beautiful aquatic panorama of Hampton Roads and Chesapeake Bay, all the sports, and a multitude of tourist attractions which include the shipyard, the Langley Field Air Base, Fortress Monroe, the Mariner's Museum and, only a short run up the Peninsula, the historic shrines of Yorktown, Williamsburg and Jamestown. So put it on your calendar—annual meeting of the Medical Society of Virginia, Chamberlin Hotel, Old Point Comfort, October 9 to 12, 1949.

CHESTER D. BRADLEY, M.D.

Scientific Exhibits

Very excellent and ample space for the Scientific Exhibits at the Chamberlain has been assigned to the Exhibit Committee. The same decorating firm which furnished the booths last year will again service us. Those who saw the exhibits last year will remember the same equipment is furnished as is used at the A. M. A. Viewing boxes for transparencies are also furnished which are fitted with fluorescent lights.

In order to secure desirable space, all exhibitors are requested to write the Chairman of the Scientific Exhibit Committee, Vincent W. Archer, Box 1637, University Station, Charlottesville, for ap-

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plication blanks as soon as possible. This will enable the committee to "block out" the space more efficiently.

Medical College of Virginia.

The Commencement Exercises of the One Hundred Twelfth Session of the College was held on June 7th at The Mosque in Richmond. The Commencement Address was delivered by the Honorable William Munford Tuck, Governor of Virginia. The Honorary Degree of Doctor of Science was presented Dr. Wortley Fuller Rudd, Dean Emeritus, School of Pharmacy of the College.

Alumni Day was held on June 6th. The following officers were elected: Dr. Alan J. Chenery, Washington, D. C., succeeded to the presidency; Dr. Donald S. Daniel, Richmond, president-elect; Drs. Alex. F. Robertson, Staunton, and Alex Haller, Wytheville, W. E. Lock, Miss Julian D. Watkins, and Claiborne Robins, all of Richmond, vice-presidents; and Dr. Harvey B. Haag, Richmond, treasurer. Dr. H. Hudnall Ware, Richmond, was among those elected to the board of trustees. Dr. Jack W. Witten, Tazewell, and Dr. William F. Mercer, Richmond, were honored by the Alumni Association. Dr. Mercer is the third oldest living alumnus of the College, having graduated in 1882.

There were sixty-eight graduates in medicine; thirty-one in dentistry; thirty-eight for bachelor of science in pharmacy; thirteen bachelor of science in nursing; nine bachelor of science in nursing education; thirty-six in nursing; and thirty-eight in physical therapy. The following are graduates in medicine with their hospital appointments:

MEDICAL COLLEGE OF VIRGINIA HOSPITALS, Richmond: Drs. Leo Blank, Hartford, Conn.; Eleanor Emily Jane Bundy, Decatur, Ga.; Wiley Holt Cozart, Fuquay Springs, N. C.; Edmund Noel Gouldin, Tappahannock; Fleta Adelaide Gregory, Norfolk; Samuel Hellerman, West Hartford, Conn.; Herbert Chester Hoover, Williamson, W. Va.; Robert Aaron Jackson, Wyliesburg; Beverly Jones, Charlottesville; William Cecil Link, Bloomington, Ind.; James Gilbert Lyerly, Jacksonville, Fla.; William Bigbie Moncure, Radford; Dillard McCary Sholes, Jr., Fredericksburg; Clarence Vincent Townsend, Inwood, W. Va.; Robert Klaus Waller, New York. N. Y.; and Sarah Lou Warren, Prospect Hill, N. C.

STUART CIRCLE HOSPITAL, Richmond: Dr. William Cary Hancock, Richmond.

JOHNSTON-WILLIS HOSPITAL, Richmond: Dr. John Pearl Ray, Jr., Norfolk.

NORFOLK GENERAL HOSPITAL, Norfolk; Dr. Benjamin Arthur Hubbard, Jr., Norfolk.

RIVERSIDE HOSPITAL Newport News: Dr. Edwin Herman Gray, Douglas, Wash.

UNIVERSITY OF VIRGINIA HOSPITAL, Charlottesville: Dr. Lilliane Ferrer Pinero, Arecibo, P. R.

UNION MEMORIAL HOSPITAL, Baltimore, Md.: Drs. Ruth Margaret Allen, Norfolk; and Alice Virginia Thorpe, Richmond.

ST. ELIZABETH'S HOSPITAL, Washington, D. C.: Dr. Virginia Claire Baldwin, Atlee.

CENTRAL DISPENSARY AND EMERGENCY HOSPITAL, Washington, D. C.: Dr. Frederick Mosby Williams, Virginia Beach.

PRESBYTERIAN HOSPITAL, Chicago, Ill.: Dr. Arnold Lanehart Brown, Jr., Elkhart, Ind.

MICHAEL REESE HOSPITAL, Chicago, Ill.: Dr. Harold Oliver Kamons, Parkersburg, W. Va.

GEORGE F. GEISINGER MEMORIAL HOSPITAL, Danville, Pa.: Dr. Francis Jones Dillard, Cartersville.

WESTERN PENNSYLVANIA HOSPITAL, Pittsburgh, Pa.: Dr. Joseph Geza Doboy, Central City, Pa.

LANCASTER GENERAL HOSPITAL, Lancaster, Pa.: Dr. Ulric Joachim Laquer, Philadelphia, Pa.

PHILADELPHIA GENERAL HOSPITAL, Philadelphia, Pa.: Dr. Philip London, Atlantic City, N. J.

SEWICKLEY VALLEY HOSPITAL, Sewickley, Pa.: Dr. Carl Elmer Stahl, Pittsburgh, Pa.

ROBERT PACKER HOSPITAL, Sayre Pa.: Dr. Ralph John Stalter, Pittsburgh, Pa.

LANKENAU HOSPITAL, Philadelphia, Pa.: Dr. Robert George Stineman, St. Davids, Pa.

BRYN MAWR HOSPITAL, Bryn Mawr, Pa.: Dr. Ralph Burnley Winston, Norfolk.

BRIDGEPORT HOSPITAL, Bridgeport, Conn.: Dr. Robert Edward Dutton, Jr., Danbury, Conn.

ST. FRANCIS HOSPITAL, Hartford, Conn.: Dr. William Warren Walthall, Jr., Richmond.

CHARITY HOSPITAL, New Orleans, La.: Dr. Hilda Garcia de la Noceda, Rio Piedras, P. R.

STATE OF WISCONSIN GENERAL HOSPITAL, Madison, Wis.: Drs. Francisco Gonzalez, Caracas, Venezuela, and Charles Gwyn Thompson, Marion.

HENRY FORD HOSPITAL, Detroit, Mich.: Dr. Edward William Green, Portsmouth.

EDWARD W. SPARROW HOSPITAL, Lansing, Mich.: Dr. Frank Joseph Sykes, Berkley, Mich.

CINCINNATI GENERAL HOSPITAL, Cincinnati, Ohio.: Dr. Ralph Edwards Haynes, Freeman, W. Va.

PRESBYTERIAN HOSPITAL, Denver, Col.: Dr. Robert Earl Holzgrafe, Evansville, Ind.

CHARLESTON GENERAL HOSPITAL, Charleston, W. Va.: Dr. George Thompson Hoylman, Buckhannon, W. Va.

OHIO VALLEY GENERAL HOSPITAL, Wheeling, W. Va.: Dr. Richard Samuel Stephens, Bluefield, W. Va.

ST. MARY'S HOSPITAL, Huntington, W. Va.: Dr. Harry Nenni, Huntington, W. Va.

JOHN SEALY HOSPITAL, Galveston, Tex.: Dr. Jacquelyn Marie Kirk, Pineville, W. Va.

BETH ISRAEL HOSPITAL, Newark, N. J.: Dr. Morton Kurtz, New York, N. Y.

VANDERBILT UNIVERSITY HOSPITAL, Nashville, Tenn.: Dr. Robert E. McClellan, Bristol, Tenn.

METHODIST HOSPITAL, Indianapolis, Ind.: Dr. John Hoover Moon, Bluffton, Ind.

MARY FLETCHER HOSPITAL, Burlington, Vt.: Dr. David Shute Palmstrom, Needham, Mass.

JAMES WALKER MEMORIAL HOSPITAL, Wilmington, N. C.: Dr. Irving Everet Shafer, Jr., Salisbury, N. C.

CITY MEMORIAL HOSPITAL, Winston-Salem, N. C.: Dr. William Thomas Walker, Brown Summit, N. C.

MORRISANIA CITY HOSPITAL, New York, N. Y.: Dr. Stanley Simon, New York, N. Y.

MARY HITCHCOCK MEMORIAL HOSPITAL, Hanover, N. H.: Dr. William Overton Winston, Norfolk.

U. S. ARMY HOSPITAL INTERNSHIPS

WALTER REED GENERAL HOSPITAL, Washington, D. C.: Drs. Martin Samuel Goldfarb, Charleston, W. Va.; Joseph Henry Masters, Newport News, and Stanley Newman, Cleveland Ohio.

U. S. NAVAL HOSPITAL INTERNSHIPS

U. S. NAVAL HOSPITAL, Bethesda, Md.: Dr. Suzanne Williamson Brown, New Market.

U. S. NAVAL HOSPITAL, Great Lakes, Ill.: Drs. Robert Thor Melgaard, Concord, Tenn.; and Mary Weatherly Tilden, Jamestown, N. C.

U. S. NAVAL HOSPITAL, Philadelphia, Pa.: Drs. Heth Owen, Jr., Richmond; and John Lemuel Thornton, Warrenton.

U. S. NAVAL HOSPITAL, San Diego, Calif.: Dr. Robert Stephen Turner, Jr., Roanoke.

U. S. MARINE HOSPITAL INTERNSHIPS

U. S. MARINE HOSPITAL, Norfolk: Dr. Donal Snellings Parker, Bedford.

U. S. MARINE HOSPITAL, Seattle, Wash.: Dr. Norman William Pinschmidt, Richmond.

Dr. Sidney Shields Whitaker, Jr., Bristol, Tenn., was also a member of this graduating class.

Dr. Coleman Honored.

Dr. Claude C. Coleman of Richmond, pioneer neurological surgeon in Virginia and the South, was honored on May 30th when thirteen prominent neurological surgeons from various sections of the nation were hosts at a celebration in his honor. These doctors all trained under Dr. Coleman at the Medical College of Virginia and are as follows: Drs. Alexander C. Johnson, Great Falls, Montana; James L. Thomson, Norfolk; J. G. Lylerly, Jacksonville, Florida; Archer A. Wilson, Charleston, W. Va.; Carroll A. Brown, Sioux City, Iowa; Charles E. Troland, Richmond; W. Gayle Crutchfield, Charlottesville; Edmund J. Morrissey, San Francisco, California; Frank H. Mayfield, Cincinnati, Ohio; Everett G. Grantham, Louisville, Kentucky; Jack Ulmer, James Walker, and John Meredith, all of Richmond. Two other hosts, who are general surgeons, but also trained under Dr. Meredith, are Drs. J. M. Emmett, Clifton Forge, and R. J. Wilkinson, Huntington, W. Va. The special occasion for this celebration was to commemorate the founding of the department of neurological surgery at the Medical College of Virginia thirty years ago with Dr. Coleman as the founder.

Technical papers were presented during the day and at night about seventy persons attended a dinner at which Dr. Coleman was presented with a silver platter engraved with the names of the 15 hosts. Among the visitors at the dinner were the following neurologists: Dr. Howard A. Naffziger, University of California, San Francisco; Dr. Gilbert Horrax, Lahey Clinic, Boston; Dr. Winchell McK. Craig, Mayo Clinic, Rochester; Dr. R. Glen Spurling, University of Louisville, Kentucky; and Dr. Charles Bagley, Jr., University of Maryland, Baltimore.

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The Base Hospital No. 45 Veterans Association

Met at the McGuire Clinic, Richmond, on May 30th for their twenty-ninth reunion. Three memorial windows dedicated to deceased members of the Association were presented. They are in memory of Dr. Stuart McGuire, commanding officer of the hospital unit and one time head of McGuire Clinic; Dr. William Lowndes Peple, chief of surgical services in the base hospital; and Dr. J. Garnett Nelson, chief of medical services in the unit. The windows are installed in the clinic library. At the business session, Mrs. Stuart McGuire was elected commander of the Association; Dr. John Bell Williams, vice-commander; Senator Lloyd C. Bird, re-elected adjutant; Mr. Thomas C. Boushall re-elected finance officer, and Mr. Robert P. Quarles was re-elected assistant to the commander and the adjutant.

Dr. Edwin P. Lehman,

Charlottesville, has been elected to membership in the Raven Society of the University of Virginia. This is one of the highest honors awarded a University of Virginia student, alumnus or faculty member.

Dr. H. Hudnall Ware, Jr.,

Richmond, was recently honored by being made a member of Omicron Delta Kappa and Mortar Board, national honorary leadership society, at the College of William and Mary.

Health Units Extended.

Extension of local health units to cover nine new localities, effective July 1st, has been announced by Dr. L. J. Roper, State Health Commissioner. These offices will serve the counties of Bedford, Franklin, Greene, Madison, Fluvanna, Louisa, Frederick, Lunenburg, and the city of Winchester. There has been a realignment of medical division supervision to take care of the newcomers, and the following localities will be under these doctors: Dr. E. C. Gates, Amelia, Chesterfield and Powhatan; Dr. S. S. Shouse, Appomattox, Buckingham and Cumberland; Dr. W. P. Jackson, Bedford and Franklin; Dr. W. W. Hargrave, Campbell and Charlotte; Dr. C. C. Kress, Culpeper, Greene, Madison and Orange; Dr. H. M. Snead, Fluvanna, Goochland and Louisa; Dr. J. W. Still, Frederick and the City of Winchester; and Dr. H. E. Jenkins, Lunenburg, Prince Edward and Nottoway.

Dr. Carl S. Lingamfelter,

Recently of Dumbarton, is now at the Columbia-Presbyterian Medical Center, New York City, where he is taking a three year post-graduate course in dermatology.

Dr. Herbert G. Ruffin,

Class of '38, Medical College of Virginia, has located at Dumbarton where he has taken over the practice of Dr. Lingamfelter.

Dr. Richard A. Bagby,

Formerly of Richmond, is now in Tampa, Florida, where he is taking special training in ear, nose and throat in the Municipal Hospital under Dr. J. Brown Farrior. He is a graduate of the Medical College of Virginia, class of 1943.

The American Gynecological Society

Held its annual meeting at Hot Springs, May 9-10. Dr. Joseph Baer, Chicago, was named president, and Dr. Norman F. Miller, Ann Arbor, was re-elected secretary. The 1950 meeting of this Society will be held at the Greenbrier, White Sulphur Springs.

Dr. Eller to Resign.

Dr. C. Howe Eller, Director of Public Health of the City of Richmond, has resigned, effective August 15th, to accept a position as health director for Louisville and Jefferson County, Kentucky. In his new position, he will be chairman of the department of preventive medicine and public health at the University of Louisville Medical School. Dr. Eller has been Director of Public Health in Richmond since 1946.

Virginia Doctors Attend Meeting.

The American Psychiatric Association met in Montreal, Canada, May 23-27, and the following Virginia doctors were registered: Drs. Rex Blankinship, J. E. Barrett, R. Finley Gayle, E. C. Hoff, Benedict Nagler, J. Asa Shield, and I. S. Zfass, all of Richmond; H. C. Davis, Granville Jones, and E. Beamer-Maxwell, Williamsburg; J. B. Pettis, S. C. Runkle, and G. E. Stone, Staunton; J. P. King and T. E. Painter, Radford; Frank A. Strickler, Roanoke; David C. Wilson, Charlottesville; and Frank Redwood, Norfolk.

Dr. Charles H. Dow,

Having recently completed his training at the Gill Memorial Eye, Ear and Throat Hospital, Roa-

noke, is opening his office in Monessen, Pennsylvania. He has also recently been made a diplomate of the American Board of Otolaryngology.

Dr. E. G. Gill,

Roanoke, has recently been appointed as a member of the National Committee for the Study of Relationship of Poliomyelitis and Tonsillectomy. The chairman of this committee is Dr. Daniel S. Cunning of New York and he has appointed one member from each state to serve with him.

Dr. Gill has also been appointed as chairman of the Committee to Study the Effects of Air Pollution Upon the Respiratory Tract and from a Health Hazard Standpoint in General. This is a committee of the American Bronchoscopic Society.

Ex-Interns of St. Elizabeth's.

The annual meetings of the Ex-Interns' Association of St. Elizabeth's Hospital, Richmond, were resumed on April 26th, with a luncheon at the Commonwealth Club. Dr. John W. Morris, of Morehead City, North Carolina, was elected Chairman for the 1950 meeting, and it was decided to hold the meeting each year on the same day of the J. Shelton Horsley Memorial Lecture at the Richmond Academy of Medicine.

American Board of Public Health and Preventive Medicine.

The following Richmond doctors have been invited to be Founders of the new American Board of Public Health and Preventive Medicine: Dr. C. Howe Eller, Director of City Health Department; Dr. E. M. Holmes, Jr., Assistant Director of City Health Department; and Dr. E. C. Harper, Veterans Administration.

McGuire Veterans Hospital—New Manager.

Dr. Leslie Hurd Wright, a native New Englander, has been named as manager of McGuire Veterans Administration Hospital, Richmond. He succeeds Dr. Edmund M. Ellerson who has transferred to the Alexandria Veterans Administration Hospital in Louisiana. Dr. Wright is a graduate in medicine of the University of Vermont and has recently been with the Wood VA Hospital in Milwaukee.

Dr. Ellerson came to McGuire early in 1946, after serving with the Army for 18 months as executive officer of the eighty-third General Hospital in England.

Married.

Dr. William Hugh Grey, Charlotte, N. C., and Miss Elizabeth Gooch Seay, Richmond, May 14th. He is a graduate of the Medical College of Virginia, class of 1948, and they will make their home in Richmond.

Dr. Glenn Claire Hall, Jr., Pulaski, and Miss Frances Elizabeth Palmer, South Boston, May 21st. He is a graduate in medicine from the University of Virginia in 1948.

Local Committees on Arrangements for Annual Meeting.

Pooling their efforts toward a common goal the Medical Societies of Elizabeth City County and Warwick County have formed a Local Committee on Arrangements for the annual meeting of the Medical Society of Virginia at Old Point Comfort, next October. The membership of the committee represents the entire medical profession of the Lower Peninsula. *Co-chairmen:* Dr. Waverly R. Payne, Newport News and Dr. Paul J. Parker, Hampton; *Entertainment:* Dr. Edward L. Alexander, Newport News, Dr. Frank A. Kearney, II, Phoebus, Dr. William A. Read, Dr. Paul Hogg, Dr. C. E. Holderby, and Dr. George G. Hankins, Newport News; *Registration:* Dr. Barnes Gillespie, Hilton Village and Dr. William A. Mitchell, Newport News; *Halls, Lanterns, Special Meetings:* Dr. Robert H. Wright, Jr., Phoebus, Dr. Nowell D. Nelms, Hampton, and Dr. Chester D. Bradley, Newport News; *Recreation:* Dr. Alfred A. Creecy, Dr. R. A. Davis, and Dr. William O. Poindexter, Newport News; *Scientific Exhibits:* Dr. J. Will Tankard, Hilton Village, and Dr. Thomas N. Hunnicutt, Newport News; *Reception:* Dr. Russell Buxton, Newport News, and Dr. Eldred S. Jones, Hampton; *Woman's Auxiliary:* Mrs. Chester D. Bradley; and *Publicity and Press Relations:* Dr. Chester D. Bradley, Newport News.

Dr. Frank A. Kearney, II is the president of the Elizabeth City County Medical Society which includes the physicians of the Hampton-Phoebus area. It is the older of the two societies since Hampton is a much older community than Newport News. The Warwick County Medical Society includes the physicians of Newport News and Hilton Village.

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Its president is Dr. George S. Grier, III of Newport News.

Dr. Hoge and Dr. Williams.

Dr. Randolph H. Hoge, Professor of Gynecology, and Dr. George Zur Williams, Professor of Pathology, Medical College of Virginia, upon invitation of the Medical Society of the State of Oregon, gave a series of lectures on cancer diagnosis and treatment in the State of Oregon, June 6-10. As a part of the extramural and postgraduate courses for the practicing physicians of Oregon, Drs. Hoge and Williams gave nine lectures. The lectures of Dr. Hoge were on the general theme of "Cancer of the Cervix and Other Gynecologic Cancer", and Dr. Williams on "General Cancers for Cancer Diagnosis and Control".

The American Council of Christian Churches,

At its spring convention in Denver, Colorado, April 27-29, unanimously adopted the following resolution against socialized medicine:

"For the State to usurp responsibility for the medical care of its citizens, whatever its name and title, constitutes an infringement of human responsibility and individual freedom which God has not given to the State.

"God created man for His own glory, gave him a body as a fit organ for his soul, and made man responsible to Him, not to the State, in his care of his body. The State has no right to destroy this relationship, and require by law, force, or other method the submission of the body to its paternal care.

"The soul and the body are inseparably connected, parted only by death; and when the State *attempts* such care of the body, it inevitably moves to direct the mind and spirit.

"The depravity of man, as taught in the Bible, so aggravates State control as it relates to government officials, politicians, physicians, and patients as to produce a corrupt, inefficient, expensive, bureaucratic and intolerable system.

"Socialized medicine in any form, represents, we believe, a clear violation of the Fourth Amendment of our Constitution which guarantees "The right of the people to be secure in their persons".

"The battle against State medicine is not for the doctors alone, but it belongs to all Christian people who cherish their own freedom as well as the physician."

Dr. C. B. Hughes

Has recently located in Wytheville, where his practice is limited to pediatrics. He is on the staff of the Chitwood-Moore Clinic. Dr. Hugh B. Brown, Jr., of Pulaski, is also now doing the x-ray work at the Chitwood-Moore Clinic.

Radford Community Hospital—Annual Clinic Day.

The Fourth Annual Clinic Day is being held by the Radford Community Hospital on July 21st. Guest speakers will be Dr. Earl O. Latimer, Assistant Professor of Surgery, Northwestern University, Chicago; and Dr. Samuel J. Fogelson, Associate Professor of Surgery, Northwestern University. Dr. Latimer will speak on Surgery of the Gall Bladder and Biliary Tract and Dr. Fogelson on The Surgical Therapy of Duodenal Ulcer.

There is usually an attendance of 100 to 125 guests at this meeting which consists of a banquet and scientific program.

The American Congress of Physical Medicine

Will hold its twenty-seventh annual scientific and clinical session Sept. 6, 7, 8, 9 and 10, 1949 inclusive, at the Netherland Plaza Hotel, Cincinnati, Ohio. Scientific and clinical sessions will be given on the days of Sept. 6, 7, 8, 9 and 10, 1949. All sessions will be open to members of the medical profession in good standing with the American Medical Association. In addition to the scientific sessions, the annual instruction courses will be held Sept. 6, 7, 8 and 9. These courses will be offered in two groups. One set of ten lectures will consist of basic subjects and attendance will be limited to physicians. One set of ten lectures will be more general in character and will be open to physicians as well as to physical therapy technicians who are registered with the American Registry of Physical Therapy Technicians. Full information may be obtained by writing to the American Congress of Physical Medicine, 30 North Michigan Avenue, Chicago 2, Illinois.

The Medical Society of the State of North Carolina

Held its Sesquicentennial Session at Pinehurst, May 9-11, under the presidency of Dr. J. F. Robertson, Wilmington. There were 998 physicians registered. Dr. G. W. Murphy, Asheville, succeeded to the presidency, and Dr. Roscoe D. McMillan, Red Springs, was named president-elect. Dr. Millard Hill, Raleigh, replaced Dr. McMillan as secretary, and Mr. J. T. Barnes was re-elected for a term of three years as executive secretary.

XUM

Medical Care Consultant Appointed.

Dr. Arthur Klein, Richmond, has been appointed as consultant in the City Health Department's new program for medical care of indigents. He will be associated with the program on a part-time basis.

U. S. Air Force Medical Service.

Organization of the United States Air Force Medical Service within the Department of the Air Force has been announced by General Hoyt S. Vandenberg, Air Force Chief of Staff. Major General Malcolm C. Grow, surgeon general of the air force, who heads the medical service, will report directly to General Vandenberg.

Highlight of the plan for the service is the provision assuring career opportunities for personnel. Housing for medical officers and their families, stability of assignment and opportunities for medical and scientific advancement are features of the plan.

"Medical specialists are assured of opportunities for advanced training in both clinical medicine and research in aviation medicine. Professional facilities of general hospitals and laboratories, approved civilian institutions and air force facilities will be used to provide regularly spaced training tours for members of the air force medical service."

The plans are designed to correct major objections of professional people to a career in the armed forces. Medical officers will be given every opportunity to pursue their specialties and doctors and dentists who volunteer to serve for more than one year will continue to receive the extra $100 a month. Officers, nurses, and enlisted technicians who qualify and are assigned flying duties will receive additional hazard pay.

Promotions and rank distributions will be administered so that personnel of all categories can begin their careers and advance through successive grades in accordance with their ability and responsibilities held.

The service will be manned by both regular and reserve officers, reserve officers having the prerogative of serving limited periods of active duty. Civilians desiring regular or reserve commissions may apply directly to the surgeon general, U.S. Air Force, Pentagon, Washington. Recognition of prior professional training and previous military service will be given in original appointments.

Doctor Wanted

To take over well equipped, eight room doctor's office, with well established practice in eastern North Carolina town of 3,000 population. Modern hospital 18 miles distant on route 301, south. If interested write Harrison Drug Company, Enfield, North Carolina. (*Adv.*)

For Sale.

Second hand Castle autoclave 18″ x 9″, complete with stand, two gallon water sterilizer and one quart still in perfect working condition. Reason for selling is need of larger autoclave. Communicate with Box 296, Wytheville, Va. (*Adv.*)

Wanted.

Used electrocardiographic recorder. Write "EC", care Virginia Medical Monthly, 1200 East Clay Street, Richmond 19. (*Adv.*)

OBITUARIES

Dr. Frank Harrell Redwood,

Prominent neuro-psychiatrist of Norfolk, died May 27th, at the age of sixty-eight. He had been an active member of the Medical Society of Virginia for thirty-four years and at the time of his death was a member of the Committee on Mental Hygiene.

At a meeting of the Norfolk County Medical Society on May 30th, the following tribute was adopted.

Dr. Frank Harrell Redwood died suddenly of coronary thrombosis on May 27, 1949, in New York City, while returning from a meeting of the American Psychiatric Society in Montreal, Canada.

A native of Nansemond County, Virginia, he had graduated in medicine at the Medical College of Virginia in 1913. Following specialization in psychiatry, he served

in the United States Army Medical Corps in World War I, and upon release from active duty, he began practice in Norfolk. He was a Diplomate of the American Board of Psychiatry and Neurology, and a Fellow of the American College of Physicians. He held membership in the American Psychiatric Society, was an organizer in the Southern Psychiatric Association, and was associated with many other medical groups. He was consultant at the United States Public Health Service Hospital at Norfolk, and had been very active as a member and Secretary of the Virginia State Board of Mental Hygiene and Hospitals. He also was an organizer of the Norfolk Mental Hygiene Clinic. All three Norfolk hospitals were fortunate in holding his interest, and he was president of the medical staffs of two of them at various times. He was also a past president of the Norfolk County Medical Society.

His wife was Miss Grace Gilman, of Richmond, Virginia, who died in June, 1946. He is survived by a brother, a son and daughter, and four grandchildren.

The medical profession and the community have lost a valuable friend and a devoted scientific worker. It is unfortunate that he should be taken at this time, when he was rendering his most valuable service.

It is requested that this humble tribute to our friend and fellow member be written into the minutes of the Norfolk County Medical Society, and that a copy be given the VIRGINIA MEDICAL MONTHLY, and a copy be sent to the family.

C. LYDON HARRELL, *Chairman*
A. BROWNLEY HODGES
WALTER P. ADAMS

Dr. Arthur Hamilton Deekens

Died in a Richmond hospital on May 29th. He had practiced in Richmond for twenty-five years but had recently been living in New Orleans. Dr. Deekens was born in Belfast, Ireland in 1865 and received his medical degree from the University of Pennsylvania in 1889. He practiced in Mexico City and New Orleans after leaving Richmond but had retired about twelve years ago because of bad health. He had been a member of the Medical Society of Virginia for thirty-six years and was made a Life Member at the 1948 meeting. Two brothers, Dr. A. B. K. Deekens of Amelia and Dr. William F. Deekens of Wytheville, survive him.

Dr. George Clegg Williams,

Well known physician of Pearisburg, died on May 29th of injuries sustained in an automobile accident which also took his father's life. He was born in Stuart and was forty-one years of age. Dr. Williams graduated in medicine from the University of Virginia in 1935. He practiced in Richlands before locating in Pearisburg in 1940 where he was plant physician for the Celanese Corporation. Dr. Williams was a vice-president and past secretary of the Southwestern Virginia Medical Society and had been a member of the Medical Society of Virginia for several years. His wife and a son survive him.

Dr. Joseph James Anderson,

Richmond, died May 19th. He was born in Louisa County and was seventy-nine years of age. Dr. Anderson graduated from the Medical College of Virginia in 1893 and had practiced in Richmond for fifty-five years, having retired recently due to ill health. He had been a member of the Medical Society of Virginia for forty-four years and was made a Life Member at the 1948 meeting. His wife survives him.

Dr. George Blight Harrison,

Fredericksburg, died May 17th, following a heart attack. He was sixty-eight years of age and a graduate of the University of Maryland, class of 1905. Dr. Harrison practiced for several years at Colonial Beach before locating in Fredericksburg. He served as Captain in the Army Medical Corps during World War I, and later became associated with Dr. Frank Pratt in founding the Pratt Clinic. He had been a member of the Medical Society of Virginia for thirty-one years and was at one time president of the Northern Neck Medical Society. His wife survives him.

Dr. Ashton Harwood,

Binns Hall, died May 18th, after an illness of several weeks. He was seventy-five years of age and a graduate of the Medical College of Virginia in 1902. Dr. Harwood practiced for twenty-five years in Charles City County, then located in Lynchburg for thirteen years, returning to his former practice in 1932. He was a Mason and a former member of the Medical Society of Virginia. Three sons survive him.